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REVIEW

RECENT ADVANCES IN TROPICAL MEDICINE
ETC., ETC.

SUPPLEMENT TO THIRD REPORT
WELLCOME RESEARCH LABORATORIES
KHARTOUM

ANDREW BALFOUR, M.D.
DIRECTOR

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Map of Anglo-Egyptian Sudan

REVIEW

OF SOME OF THE

RECENT ADVANCES IN TROPICAL MEDICINE

HYGIENE AND TROPICAL VETERINARY SCIENCE, WITH SPECIAL REFERENCE TO THEIR POSSIBLE BEARING ON MEDICAL, SANITARY AND VETERINARY WORK IN THE ANGLO-EGYPTIAN SUDAN

BEING A SUPPLEMENT TO THE

THIRD REPORT OF THE

WELLCOME RESEARCH LABORATORIES

AT THE

GORDON MEMORIAL COLLEGE

KHARTOUM

BY

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PREFATORY NOTE

IT is a difficult matter for medical and veterinary officers stationed in the Sudan, especially those who happen to be in out-stations or who have to travel frequently, to keep in touch with current literature. This Review is intended to help them in some measure, to serve as a guide to new books and papers, and to present in a small compass the most important recent discoveries on the subjects indicated. It is also intended to indicate in what directions our knowledge as regards tropical and veterinary medicine, bacteriology and hygiene is deficient in the Sudan, and it is hoped that it will thus stimulate research and lead to the acquisition of useful information. References are given so that those who wish to go more fully into any special subject may be able to obtain the original book or paper. Every care has been taken to render these as correctly as possible. No attempt has been made to produce a text-book, and for the most part the references have been confined to sound practical papers likely to be helpful, but the scientific aspect of certain questions has been considered for the reasons stated above.

While in the main intended for medical and veterinary officers in the Sudan, many of whom have rendered the laboratories valuable assistance, it is hoped that workers in other tropical countries, where the conditions are similar to those obtaining in the Sudan, may find this Review of service. It is possible that it may also appeal to the students of Tropical Medicine in temperate climates, especially such as may be preparing for special examinations.

At the same time, it is to be regarded as supplementary to the Third Report of the Wellcome Research Laboratories, and hence the range of subjects dealt with is, of necessity, limited.

REVIEW

Of some of the more Recent Advances in Tropical Medicine, Hygiene and Tropical Veterinary Science, with special reference to their possible bearing on Medical, Sanitary and Veterinary work in the Anglo-Egyptian Sudan.*

Ainhum. Ashley-Emile,[1] in an interesting paper on ainhum, is inclined to trace a connection between ainhum and leprosy, regarding the former as a modified expression of the latter in persons of a "leprous diathesis." His argument is rather laboured, but there may be something in the anatomical reasons he advances for the seat of election of the disease. He believes the flexor tendon of the small toe to be specially subject to strain during the act of carrying heavy burdens, and that this, combined with an enfeebled nerve supply, leads to fibroid degeneration round the joint with resulting occlusion of arteries and strangulation of the toe, which enlarges owing to venous dilatation.

Wellman,[2] on the other hand, adduces evidence to show that ainhum and leprosy are not related, and in a later paper suggests that the chigger may play an important part in the development of the complaint. He points out that this theory accounts in large measure for the geographical distribution of the disease. Apart from these theories, ainhum has been stated to be due to injury, to be a trophoneurosis, a circumscribed scleroderma, a congenital, spontaneous amputation, and the result of self-mutilation by ligatures, wearing of toe-rings, etc.

Manson[3] favours the traumatic theory, and cites a similar condition affecting the tail of a pet monkey. There is nothing new to record regarding treatment. Ainhum occurs in the Sudan, and I have seen an imported case in Khartoum.

So far as is at present known, the chigger is confined to the Bahr-El-Ghazal Province, while Dr. Wenyon reports ainhum to be common at Bor on the White Nile. The natives attribute the condition to injury caused by the coarse grass. It would be interesting to determine accurately if the distribution of the disease and of the chigger coincide in the Southern Sudan.

Air. The remarkable influence of rain as a purifier of the atmosphere was well shown by an investigation[4] carried out in London in the summer of 1903. A rainfall of about 3·8 inches in five days actually was responsible for the removal of 3738 tons of solid impurities. Of these no less than 2000 tons consisted of soot and suspended matter, common salt and sulphate of ammonia constituting the remainder.

This does not take into account the great bacterial purification also effected.

Much of the Northern Sudan is practically rainless, and there can be no doubt we suffer from the lack of the freshening effect of rain upon the atmosphere. This, as has been pointed out, is due possibly to an oxidising action and perhaps to the formation of peroxide of hydrogen. No one who has lived long in Khartoum but knows there are times when the air seems lifeless and heavy. Indeed, this is frequently the case in the late afternoons in the winter. Doubtless the feeling is in part due to the dying down of the breeze, but though the air is free from gross impurities it is charged with organisms, especially with moulds.

In this connection allusion may be made to Gordon's[5]† work on the presence of streptococcus brevis in the saliva, and its use as an indicator of air pollution. By this means he has shown the presence of particles of saliva in the air at a distance of 40 feet in front of a speaker. It would be interesting to know if conditions differ greatly in a hot,

---

* With the exception of the article on Typhus Fever, the notes referring to the Sudan and a few other paragraphs, the portion of the Review from "Tuberculosis" onwards is the work of Mr. R. G. Archibald.

The Review only extends to papers, etc., appearing in journals not later than about the middle of July, 1908.

[1] Ashley-Emile, L. E. (February 1st, 1905), "On the Etiology of Ainhum." *Journal of Tropical Medicine*, p. 33.

[2] Wellman, F. C. (October 2nd, 1905), "Ainhum and Leprosy, a Critical Note." *Journal of Tropical Medicine*, p. 285.

[3] Manson, Sir Patrick, London, "Tropical Diseases." 4th Edition. 1907.

[4] "Some Interesting Facts Regarding the Purifying Effect on the Air of the Recent Rain." *Lancet*, p. 1759, Vol. I. June 20th, 1903.

[5] Gordon, M. H., Report of Medical Officer Local Government Board, 1902-1903.

† Article not consulted in the original.

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Ankylostomiasis— *continued*

As regards the larvæ of *Necator americanus* (*Uncinaria americana*), Smith[1]* has shown that they produce a substance which is very irritating to the skin and leads to severe itching with a tendency to vesiculation. This irritation leads to scratching, and the latter may actually facilitate the passage of the larvæ through the skin.

Leiper,[2] who employs the term *Agchylostomiasis*, has shown that the so-called "American" Hook-worm is widely distributed in Africa. It occurs on the West Coast, in Uganda and in North-West Rhodesia, but apparently not in Egypt. It has also been found in Ceylon, Assam and Burma, and probably is world-wide.

I cannot speak to its presence in the Sudan. The eggs of what is apparently *A. duodenale* are frequently found in the stools of Egyptian soldiers in Khartoum, but one has not had time to work at this subject. The disease is certainly not so much in evidence as in Egypt, but there seems no reason why it should not occur and spread in the Sudan, though probably Egyptians are more liable to infection than Arabs or Sudanese.

Sandwith[3] indeed notes its prevalence in Upper Egypt and suggests that it will increase as irrigation increases. I think this is very likely, and that measures should be taken to guard the Sudan, as far as possible, from its invasion. This is admittedly a very difficult matter at the present time, but seems worthy of consideration. The careful medical inspection of recruits in Egypt no doubt weeds out a considerable number of advanced cases, while up to the present there has been very little, if any, immigration of the Egyptian fellaheen into the Sudan for purposes of agricultural work. Possibly this may change in the future and then it would certainly be advisable to have some system of medical examination and to either reject infected individuals or submit them to suitable treatment before admission to the Sudan. In this connection one may[4]* note the remarkable results obtained in the campaign against ankylostomiasis in Porto Rico. The overwhelming importance of the disease as a factor in the industrial efficiency of that island having been fully established, steps were taken to treat the infected with thymol, partly in hospital but mostly as dispensary out-patients. Beta-naphthol, which is much cheaper, was also tried, but was not nearly as good in the case of out-patients. Nearly 20,000 persons were treated within a period of six months and with most gratifying results. It was found that, as a rule, five doses of thymol were sufficient to practically cure a patient, *i.e.* to render him healthy and to reduce his power, by nineteen-twentieths, of infecting the soil afresh. The report is well worth perusal, and the cost of the operations was remarkably low. It is evident that a great deal can be done by energy and persistence, even in a country sorely stricken by this most debilitating and frequently fatal disease.

Hermann's method of treatment by means of eucalyptus oil, chloroform and castor oil was described by Philipps,[5] who recorded good results obtained with it in Cairo. It seems to be efficient and practically free from danger.

For the ankylostomiasis of mines, sodium chloride has been shown to be a prophylactic[6] and can be used in a 2 per cent. solution as a spray, but, as Looss points out in a paper[7]* dealing with many particulars of the life-history of the larvæ, the only efficient preventive measure is an efficient system of conservancy. Hence the necessity of arranging for such, especially in cultivated portions of the Northern Sudan where moist conditions of the soil prevail and where, if this sanitary measure be neglected, the disease may establish itself and in the future produce much invaliding and incapacity for work. It is worth noting, however, that some hold the view, a view not shared by Looss, that the larvæ show remarkable powers of resisting dryness, so that even under ordinary conditions in the Sudan there may be danger from the employment of imperfect conservancy methods which permit systematic fouling of the surface soil of towns and villages.

---

[1] Smith, C. A. (November 24th, 1906). *Journal of American Medical Association.*

[2] Leiper, R. T. (March 23rd, 1907), "Distribution of American Hookworm." *British Medical Journal*, p. 683, Vol. I.

[3] Sandwith, F. M., "Medical Diseases of Egypt," Part I., 1905.

[4] Prelim. Report of the Comm. for the Suppression of Ankylostomiasis in Porto Rico, San Juan, December 31st, 1905.

[5] Philipps, L. P. (December 1st, 1905), "On Eucalyptus Oil as a vermifuge in Ankylostomiasis." *Journal of Tropical Medicine*, p. 341, Vol. VIII.

[6] Manouvriz, A. (November 25th, 1905), "The Prophylaxis of Ankylostomiasis." *British Medical Journal*, p. 1418, Vol. I.

[7] *Zeitschrift. für Klin. Med.*, t. LVIII., p. 43.

* Article not consulted in the original.

Bacteriology —*continued*

sources. Trillat, quoted by Arnold, regards the action as due to the presence of formaldehyde in tobacco smoke. The kind of tobacco employed seemed to exert no influence on the results.

Castellani[1] has isolated from the blood of three patients suffering from fever, in Ceylon, an organism which he has called *Bacillus ceylanensis*. Further particulars will be found under the heading of "Fever" (*page* 69), but it may be stated here that the bacillus was non-motile, produced a pellicle in broth, acidified and coagulated milk slowly, produced acid but no gas in glucose, and produced neither acid nor gas in saccharose, mannite, dulcite or lactose. There was no indol formation. From a fourth case, in which the clinical symptoms were slightly different, a somewhat similar organism was isolated, but it acidified and clotted milk quickly and formed indol. In every instance the organisms were agglutinated by the blood of the patients from whom they were recovered.

When examining a well-water in Khartoum, I came across an organism which morphologically and culturally resembled this *B. ceylanensis*, but the only sugar media in which it was tested were lactose and glucose and its pathogenicity was not determined. Intraperitoneal injections of broth cultures of *B. ceylanensis* killed guinea pigs in 24 to 36 hours.

Buckley,[2] in a very important paper, records the results of his careful and elaborate experiments on the resistance of some pathogenetic micro-organisms to drying. Those used were *Staphylococcus pyogenes aureus*, *Bacillus coli communis*, *Bacillus typhosus*, *Bacillus diphtheriæ*, *Bacillus pestis* and *Spirillum choleræ*. Of these, *Staphylococcus pyogenes aureus* was found to be the most, and *Spirillum choleræ* the least, resistant.

The latter cannot live in a condition of complete dryness. Of the other four, *B. coli communis* and *B. typhosus* proved more resistant than the *B. diphtheriæ* and *B. pestis*. The remaining conclusions I quote verbatim:—

Some organisms live longer in a moist and others in a dry atmosphere. In the first class are the *Spirillum choleræ* and the *Bacillus coli communis*, which live very much longer, and the *Bacillus typhosus* and the *Bacillus pestis*, which live only slightly longer, in a moist atmosphere than in a dry one. In the second class are the *Staphylococcus pyogenes aureus* and the *Bacillus diphtheriæ*.

Speaking generally, the absolutely dry atmosphere of the desiccator is less harmful to the bacteria used in these experiments than the partially dry atmosphere of the room. This is possibly due, as suggested by some observers, to the quick drying of the outer portions of the individual bacilli, which would result in the formation of a complete protective coat for each organism. The cholera spirillum is an exception to this rule.

The material infected exerts a considerable influence on the powers of resistance to drying possessed by the different organisms; but this influence is not of the same kind on all bacteria nor under all conditions of dryness or moisture. On examining the Tables it will be found that the longest life was reached usually on plaster and lime wood. The single exception is in the case of the *Bacillus pestis*, which was very short-lived on lime wood, and this was the case in each of ten series of experiments. All the organisms were short-lived on paper. As would be expected from the fact that the emulsion is unable to sink into glass, and would consequently dry rapidly, the organisms did not live very long on that material.

The effect of pine wood was variable, and especially so in the moist chamber, pointing to the fact that some constituent or constituents of the wood were capable of acting injuriously upon the organisms in the presence of moisture. In all cases this variety of wood exercised an adverse influence on the organisms, and this suggests the advisability, from a sanitary standpoint, of the use of pine wood, as far as possible, in such buildings as hospitals—and especially hospitals for infectious diseases.

Infection can persist in dry buildings, cloths, etc., for at least the following periods:—

Staphylococcus pyogenes aureus	for 140 days.
Bacillus diphtheriæ	„ 114 „
Bacillus coli communis	„ 92 „
Bacillus typhosus	„ 91 „
Bacillus pestis	„ 34 „
Spirillum choleræ	„ 12 hours.

(These figures represent in each case the longest period during which the organism was found living on any material in the desiccator, or in the air of the room).

In the case of certain organisms, infection may persist for even longer periods if the buildings, etc., are damp:—

Bacillus coli communis	for 168 days.
Bacillus typhosus	„ 119 „
Bacillus pestis	„ 45 „
Spirillum choleræ	„ 21 „

[1] Castellani, A. (January, 1907), "Notes on cases of Fever frequently confounded with Typhoid and Malaria in the Tropics." *Journal of Hygiene*, p. 1, Vol. VII.

[2] Buckley, G. G. (February, 1907), "The Resistance of some Pathogenic Micro-organisms to Drying." *Public Health*, p. 290.

Of very great interest to us in the Sudan are the epidemiological instances cited in connection with Enteric Fever. These are as follows:— Bacteriolog —continu

Henrot (quoted in the *Lancet*, 1896, 1901, 1903, 1907) gives particulars of an epidemic of typhoid fever which occurred in two regiments of cavalry quartered at Rheims. During some manœuvres the men rode over ground which had been manured with night-soil. The weather was dry, and much dust was produced, which was inspired and swallowed by the troops. A bad smell was noticed at the time. Shortly afterwards the epidemic broke out amongst these men. The water supply was not to blame, as other people drinking from the same source were not affected.

In the *British Medical Journal* of November 10th, 1900, is an article on the outbreak of typhoid fever at Quetta, India. At this place the water supply is derived from the hills and was above suspicion. There was, as usual, freedom from typhoid fever up to May, but from May 2nd to 13th there were dust storms. Sore throats and tonsillitis resulted, followed by an outbreak of typhoid fever, some of the cases commencing with sore throat. The night-soil was placed in pits to the north-west, from which quarter the prevailing winds blew, and in the dry air the deposited matters were dried and blown about as dust. Those companies suffered most who were nearest to the filth pits. The air coming from the direction of the pits contained "large numbers of germs that are invariably present in fæcal matter, and not in pure air, although the enteric bacillus itself was not isolated." It appears, from a subsequent article in the issue of September 14th, 1901, that the outbreak ceased on the removal of this source of infection.

The foregoing examples, which could easily be multiplied, will suffice to indicate the probable influence of desiccated products on the dissemination of disease.

It is a pity that no reference is made to experiments with the bacillus of dysentery, though this subject will be discussed in its proper place. It would be useful also to have reliable data dealing with the combined influence of drying and high soil and atmospheric temperature.

The bacteriology of the common cold has been the subject of considerable work and discussion. Miller[1] confirmed work of earlier observers by showing that the organisms found in catarrh are those normally present in the nasal mucous membrane. These were chiefly *Staphylococcus pyogenes albus*, *Streptococcus pyogenes* and an undetermined *diplococcus*. *Staphylococcus pyogenes aureus*, Hoffmann's bacillus and *Bacillus subtilis* were also found. He points out the mechanism of infection, *i.e.* the chill, resulting lowered vitality of the mucous membrane, disturbed balance between the tissues and the germs, bacterial action and the reaction of the tissues leading to the presence of leucocytes and antitropic bodies. Treatment can only be successful in the incubation period, usually of 24 hours' duration, and is to be sought in the inhalation of a volatile antiseptic such as eucalyptus oil.

Benham,[2] as the result of a specially careful investigation, found in a series of 27 cases both *Diphtheroid bacilli* and the *Micrococcus catarrhalis* which was isolated by Hajek. He thinks the former may be responsible rather for painful sore throat with headache, malaise and muscular pains, irritable cough and scanty, viscid expectoration, than for the true coryza symptoms. He suggests it be called *Bacillus septus*, or "Cautley's" bacillus, after its discoverer, who named it *B. coryzæ segmentosus*. Pfeiffer's bacillus was scarcely in evidence at all, a point of considerable interest.

Allen,[3] while pointing out that several organisms are operative, presses the claims of Friedlander's bacillus and adduces evidence in its favour as being of etiological importance, at least in the type of cold characterised by shivering, general depression and malaise, with acute running from the nose and eyes. He admits that *Diphtheroid bacilli* may play a part in cases with sore throat, cough and scanty, viscid expectoration.

Gordon[4] suggests that animal experiments might help to settle the question. In a review[5] of the whole subject we find that, in 50 out of 56 cases examined, the *B. coryzæ*

[1] Miller, J. (May, 1906), "The Etiology of Coryza." *Birmingham Medical Review.*

[2] Benham, Chas. H. (May 6th, 1906), "The Bacteriology of a Common Cold." *British Medical Journal*, p. 1023, Vol. I.

[3] Allen, R. W. (May 12th, 1906), "The Bacteriology of a Common Cold." *British Medical Journal*, p. 1131, Vol. I.

[4] Gordon, M. H. (June 2nd, 1906), "The Bacteriology of a Common Cold." *British Medical Journal*, p. 1193, Vol. I.

[5] Gordon, M. H. (September 22nd, 1906), "The Bacteriology of a Common Cold." *British Medical Journal*, p. 1318, Vol. I.

Bacteriology —continued

segmentosus was present but its precise etiological significance in relation to the common cold has yet to be determined. The *Micrococcus catarrhalis* has more recently been the subject of work by Arkwright,[1] whose conclusions are as follows:—

1. Gram-negative cocci derived from the nose can be divided into several different races, which require careful culture for their identification.

2. *M. catarrhalis* is present very frequently in the normal nose, especially in the young and more especially in infants.

3. Its frequency does not appear to be greater in ordinary catarrhal states than in non-catarrhal. In this respect it differs from the *pneumococcus* and Hoffmann's bacillus.

It would, I think, be interesting if some work on this subject were carried out in tropical countries. In the Sudan, at certain seasons of the year, the influenzal type of cold is very common, the exciting causes being chill, and, to a lesser extent, the irritation produced by dust. It is possible that diphtheroid organisms are not so prevalent as amongst urban populations in temperate countries, and it might be possible to settle which is the true organism of coryza more easily than in a place where micro-organisms abound and conditions are more complicated.

It is worth noting that spraying the floors of schoolrooms with weak formalin solution has been found to inhibit the spread of infectious colds.

Gwyn and Harris[2] have worked out a comparison between the results of blood cultures taken during life and after death, a subject of very considerable importance. Their chief conclusions are as follows:—

1. That within certain limits post mortem bacteriological methods afford trustworthy means of determining or confirming the presence of any of the well-known infectious processes.

2. That the more often a marked ante mortem infection is present, the greater probability there is of finding an uncomplicated post mortem bacteriological result, provided the investigation is carried on within a reasonable time after death, say 12 hours.

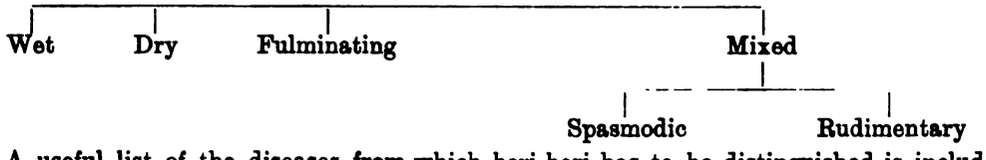
3. That a bacteræmia due to the common organisms of the intestinal tract and the so-called "agonal invasions" of the blood streams do occur, yet they should not be assumed to be present with any great degree of frequency.

With regard to 2, one can say from the result of animal post mortems that the period mentioned has to be greatly shortened for the Sudan. In the summer a period of four hours, as a rule and under ordinary conditions, suffices for the occurrence of a general bacteriological infection which is especially fatal to examinations having for their object the recovery of hæmatozoa from the blood and organs.

But little is said in text-books regarding the important ultra-visible viruses which are of special interest from the veterinary standpoint. A paper on this subject by Macfadyean[3] is, therefore, likely to prove of great value. In the first instalment he deals with the viruses of the mosaic or spotted disease of the tobacco plant, of foot and mouth disease, and of African horse-sickness and also discusses the technique employed for the isolation of the ultra-visible viruses.

Beri-beri. The vexed question of the precise etiological factor determining this disease still remains unsettled, and this despite a great deal of work by able observers. It is impossible to detail at all fully the various researches and their results, but mention may be made of the essay by Gerrard,[4] which gives a very graphic description of the symptoms.

He appends a table of types as follows:—



A useful list of the diseases from which beri-beri has to be distinguished is included. These are alcoholic neuritis, arsenical neuritis, lead-poisoning, malarial cachexia, Landry's

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[1] Arkwright, J. A. (January, 1907), "On the occurrence of the *Micrococcus catarrhalis* in normal and catarrhal noses, and its differentiation from other Gram-negative cocci." *Journal of Hygiene*, p. 145, Vol. VII.

[2] Gwyn, N. B., and Harris, N. MacL. (June, 1905), "A Comparison between the results of blood cultures taken during life and after death." *Journal of Infectious Diseases*, p. 514.

[3] Macfadyean, J. (March, 1908), "The Ultra-visible Viruses." *Journal of Comparative Pathology and Therapeutics*, Vol. XXI., Part I.

[4] Gerrard, P. N., "Beri-beri, its Symptoms and Symptomatic Treatment." London, Churchill.

ascending spinal paralysis, locomotor ataxy, ataxic paraplegia, spastic paraplegia, myelitis affecting the lumbar region, epidemic dropsy, pernicious anæmia, heart-disease, Bright's disease, ankylostomiasis, trichinosis, pellagra, ergotism and lathyrism. It is curious that in this long list scurvy is not included, for the resemblance of certain cases of scurvy to certain types of beri-beri is well known. **Beri-beri—** *continued*

Thus, Barnardo,[1] in a paper on scurvy affecting troops in Somaliland, draws attention to toxæmic cases characterised by a neuritis sometimes peripheral, sometimes cardiac, and suggests that both scurvy and beri-beri may be due to toxin production in the alimentary tract, as suggested for the latter by Hamilton Wright, an hypothesis first put forward by Chevers. Holst and Frölich[2] term ship beri-beri "the younger brother of scurvy," but point out that it is possibly a different disease from tropical beri-beri. However that may be, beri-beri has been mistaken for scurvy more than once, and their resemblance has to be kept in mind, especially when one is dealing with advanced, untreated scurvy. This fact was forcibly brought to my notice by an epidemic of the latter which occurred in the Civil Prison, Khartoum, during the winter of 1906. Several of the patients developed symptoms strongly recalling dropsical beri-beri. Hyperæsthesia, œdema, peculiar gait, cardiac dilatation and other classical signs were present, though most of them presented, in addition, spongy and bleeding gums. Epidemic dropsy was the other disease which had to be differentiated, and it was only when energetic treatment, principally of a dietetic nature, was established that the question of diagnosis was settled.

The disease is known to occur in the French Sudan, while Bagshawe[3] suggests on very strong evidence that the condition "Bihimbo," in Uganda, is really beri-beri. Hodges[4] comments on this and records his opinion that the disease is, in all probability, beri-beri. Captain Ensor states that he has seen two typical cases in the Sudan, and I have met with a case of peripheral neuritis not unlike beri-beri. As, therefore, the disease may come more into prominence in the future, a few notes on recent work upon it may prove useful. As regards its etiology, facts favouring the mouldy rice theory are quoted by Gimlette[5] in a paper on a localised outbreak in the Malay Peninsula, while, under "Current Topics," the *Indian Medical Gazette*[6] deals with the same question and cites, as does Gimlette, the suggestive work of Hose and Lucy of Penang. The theory that beri-beri is due to an intoxication by a poison conveyed in "uncured" rice originated with Braddon, who has recently published a large work[7] on the subject and brought forward such evidence that, to quote a review, "one is inclined to come to the conclusion that rice does play a part in the production of the disease, or, at least, that further experiments should be carried out to prove or disprove its action as a cause." Fletcher[8] supports Braddon's view as a result of his experience and experiments in connection with an outbreak at Kuala Lumpur Lunatic Asylum. He concludes that:—

> Uncured rice is, either directly or indirectly, a cause of beri-beri, the actual cause being either (1) a poison contained in the rice; (2) deficiency of proteid matter, the disease being due to nitrogen starvation; or (3) uncured rice does not form a sufficiently nutritive diet and renders the patient's system specially liable to invasion by a specific organism which is the cause of beri-beri.

This leads us to speak of the organismal theories and to quote Herzog,[9] whose investigations are recorded in a very complete and interesting paper. His experiments led him to believe that none of the claims brought forward for the discovery of a specific micro-organism for the disease can be looked upon as substantiated. This includes

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[1] Barnardo, J. F. (July, 1904) "Scurvy in Somaliland: Notes on the Condition of Blood Serum." *Indian Medical Gazette*, p. 241, Vol. XXXIX.

[2] Holst, A., and Frölich, T. (October, 1907), "Experimental Studies Relating to Ship Beri-beri and Scurvy." *Journal of Hygiene*, Vol. VII., No. 5.

[3] Bagshawe, A. G. (January 15th, 1907), "'Bihimbo' Disease: The Nature of the Disease termed 'Bihimbo' met with in the Chaka District of the Uganda Protectorate." *Journal of Tropical Medicine and Hygiene*, p. 18, Vol. X.

[4] Hodges, A. D. P. (October 31st, 1906), "Report to P. M. O. Uganda and East Africa on Sleeping Sickness."

[5] Gimlette, J. D. (September 1st, 1906), "Beri-beri, Mouldy Rice: The Occurrence of Beri-beri in the Sokor District."

[6] "Beri-beri and Diet." (May, 1906). *Indian Medical Gazette*, p. 183, Vol. XLI.

[7] "The Cause and Prevention of Beri-beri." London, Rebman, Ltd., 1907.

[8] Fletcher, W. (June 29th, 1907), "Rice and Beri-beri." *Lancet*, p. 1776, Vol. I.

[9] Herzog, M. (September, 1906), "Studies in Beri-beri." *Philippine Journal of Science*, p. 709, Vol. I.

Beri-beri—*continued* Hamilton Wright's[1] bacillus found in the gastro-duodenal lesions described by him, and various other bacilli and cocci which have been claimed as etiological factors, together with Glogner's amœba and the hæmatozöon put forward by Fajardo. Herzog expresses his belief that "the disease is due to an organism which gains entrance into the human body either directly or through food, and there produces a toxin which in character and effect is similar to the diphtheria or tetanus toxin, and which, by an accumulative action, gives rise to the well-characterised anatomical and histological lesions of beri-beri."

This is more or less in accord with Daniels'[2] conclusions, who regards beri-beri as an infectious disease, and points out that there is no evidence that an intermediate host is required, but that if such is required it must be a bed-bug or a flea. Indeed, he tends to think that a protozöon may yet be found.

In this connection mention must be made of the recent observations by Hewlett and de Korté[3] on a disease in monkeys closely resembling beri-beri. In the urine of these monkeys, and also in that of beri-berics, they found peculiar highly refractile bodies which they think may be protozoa. They also describe certain inflammatory changes common to the monkey's kidneys and the kidneys of a number of cases of acute beri-beri sent them from Singapore. In reply to a paper by Wright, they point out[4] that it is the intra-tubular hæmorrhages to which they specially refer. A recent review of the whole subject is that by Nocht,[5]* while experimental work has been performed by Hunter and Koch[6] in Hong Kong, who employed monkeys, and believe it is impossible to transmit beri-beri from man to animals, and that, in the strictest sense of the term, beri-beri is not an infectious disease. Other recent work is that of Tsuzuki,[7]* who has found what he calls the "kakke coccus" in the urine, stools and intestines of beri-berics, an organism which is agglutinated by the blood-serum of beri-berics and produces in animals a disease which closely resembles human beri-beri. As regards prophylaxis and therapeusis, there is little new to note. Herzog mentions that women sick with beri-beri should not nurse children, describes a method of treatment in vogue in Japan, and states that "rice should, in private practice at least, be entirely withdrawn from the daily diet of the patient."

Beverages. Under this heading attention may be drawn to the review of Dr. Hamer's[8] report on aerated waters. This shows how often waters, in themselves good, become contaminated in process of conversion to aerated waters by the use of unclean charcoal filters, by faulty storage, by faulty bottle cleaning, and especially by neglect in purifying stoppers. It also points out that the evidence regarding the ability of carbonic acid gas to destroy pathogenetic organisms, such as the *Bacillus typhosus*, is inconclusive and quite insufficient to warrant neglect of precautions.

This subject, which is one of much importance in the Sudan, will be further discussed under "Sanitary Notes" (Third Report). It is said the *Spirillum choleræ* speedily perishes in well-aerated waters, and in India[9] it is recommended that such waters be drunk when cholera is prevalent, provided no bicarbonate of soda has been added.

Attention having been drawn to the presence of antimony in bottled beverages, the poison having been derived from the rubber rings used to make the stoppers fit tightly, Thresh[10] investigated the subject. He concluded that the solubility of the antimony sulphide contained in the rubber is so slight that the only danger to be apprehended is from detached particles, and especially if old rings are used. At the same time, he notes that antimony

[1] Wright, Hamilton (May, 1902), "On the Classification of Beri-beri." *Studies from Institute for Medical Research*, Federated Malay States, Vol. II.

[2] Daniels, C. W. (1906), "Observations in the Federated Malay States on Beri-beri," Vol. IV.

[3] Hewlett, R. T., and de Korté, W. E. (July 27th, 1907), "On the Etiology and Pathological Histology of Beri-beri." *British Medical Journal*, p. 201.

[4] Hewlett, R. T., and de Korté, W. E. (November 2nd, 1907), "The Pathological Histology of Beri-beri." *British Medical Journal*, p. 1281.

[5] Nocht, B., "Real Encycl. d. gesamt. Heilkunde." 4th Edition. Berlin and Vienna.

[6] Hunter, W., and Koch, W. V. M. (November 1st, 1907), "Experimental Beri-beri in Monkeys." *Journal of Tropical Medicine and Hygiene*, p. 346. Vol. X.

[7] Tsuzuki. *Archiv für Schiffs-und Trop. Hyg.*, Bd. X., Heft 13.

[8] Hamer, W. H. (July 4th, 1903), "The Purity of Aerated Water." *Lancet*, p. 40.

[9] Duke, I., Calcutta, 1904, "The Prevention of Cholera, and its Treatment."

[10] Thresh, J. C. (November, 1905), "The Presence of Antimony in Bottled Beverages." *Public Health*, p. 95, Vol. XVIII.

* Article not consulted in the original.

sulphide is a cumulative poison and that, as the quantity in the rings is considerable, a rubber free from such poisonous ingredient should be used. **Beverages—** *continued*

A serious indictment of both tea and coffee is put forward by Fernet[1]* who calls them "satellites of alcoholism," and describes caffeism and theism in acute and chronic forms. Coffee is especially libelled, because its abuse has recently increased in France. It is said to depress the mental power, and chronic coffee intoxication leads, it is asserted, to impotence and sterility, while the children of coffee drinkers are ill-formed, ill-nourished, abnormally excitable and often suffer from arrests of development. The quantity sufficient to produce such dire results is uncertain, but three or four small cups daily may be enough to cause chronic intoxication.

These remarkable statements certainly do not find confirmation in the Sudan, where coffee is largely drunk both by Europeans and natives, though be it noted, it is very excellent coffee, prepared directly and carefully from the bean. Indeed, as the *British Medical Journal*[2] remarks: "There can be little doubt that Dr. Fernet's article is somewhat tinged by exaggeration, though it is well to bear in mind that some of these ill effects may be encountered in practice from personal idiosyncrasy or excessive use of tea and coffee."

Bilharziosis. The most recent work on this subject will be found incorporated in Madden's[3] monograph which, dealing as it does with the disease from an Egyptian standpoint, is of special interest to us in the Sudan. The author supports Looss's theory as to the direct entry of the *miracidium* by way of the skin, and he rejects Sambon's supposition that there are two species of *Schistosomum*, one characterised by terminal-spined, the other by lateral-spined, ova. He also mentions an interstitial nephritis due to the disease, and deals with its effects on the female generative organs.

Looss[4] in a recent paper severely criticises Sambon's views, and regards the evidence adduced by the latter as wholly inadequate to prove the existence of *Schistosomum mansoni*. He points out that no distinctive anatomical character of *S. mansoni* has been demonstrated, and, as regards the egg, states that proof of its belonging to a definite species must consist in showing that one form of egg is constantly connected with a certain anatomical structure, and the other form as constantly connected with another anatomical structure of the adults. "Until this is done," he says, "I am afraid that *S. mansoni* will find little approval with zoologists in spite of Dr. Sambon's contention that to zoologists the character of the ovum should suffice for the determination of a new species."

Looss goes on to say that the position of the spine depends on the relative position of the egg during the process of its formation in the ootypes, and points out that long ago Bilharz found that in Egypt the eggs of *S. hæmatobium* and *S. mansoni* may occur in one and the same individual female. Moreover, he would lay no stress on the point if a lateral-spined egg happened to be found in the urine. To him it would appear as an accidental exception, due to accidental reasons, to the rule that the urine contains terminal-spined eggs only. One important statement made is, that the lateral-spined eggs do not come from the rectal lesions. They are probably abnormal eggs, for Looss has found that very generally Trematodes, as they approach sexual maturity, form such ova. At the same time, he does not pretend that *immaturity* is the sole cause of the lateral spine. Indeed, an immature female may quite possibly produce a terminal-spined egg.

Taking up the question of geographical distribution, Looss apparently shows that Sambon's position is untenable, and mentions that in Letulle's case, where the bladder was entirely free from infection, both forms of egg were found.

One cannot follow Looss throughout his whole argument, but one statement must be noted. His experiments to find an intermediate host in various species of mollusc have invariably failed, and he has been forced to the conviction that "*Man himself acts as intermediary host.*" If this be true, then the spread of *S. hæmatobium* is not limited by the natural geographical distribution of a special intermediary host. He now believes, and he adduces some proof in favour of the idea, that the miracidia enter through the skin and that a few of them reach the liver and there form sporocysts. He proceeds to discuss this

[1] Fernet, *Semaine Médicale*, No. 31, 1906.

[2] *British Medical Journal*, p. 652, Vol. II. (September 15th, 1906), "Our Breakfast Beverages."

[3] Madden, F. C., "Bilharziosis," Cassell & Co., 1907.

[4] Looss, A. (July 1st, 1908), "What is Schistosomum Mansoni?" *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. II., No. 3.

* Article not consulted in the original.

Bilharziosis *—continued*

view at length, and states that a first infection with a female sporocyst would give a picture typical of "Manson's Bilharziosis," *i.e.* an untouched bladder, but lateral-spined eggs appearing for years in the fæces.

Whatever may be the truth regarding *S. mansoni*, and I confess that, considering Looss's vast experience and great repute as an helminthologist, his opinion carries most weight, there can be no doubt as to the importance and interest of his paper, which should be carefully studied by all interested in Bilharziosis.

A useful and well-illustrated paper is that of Sandwith,[1] who mentions Dight's suggestion to inject large quantities of sulphuretted hydrogen and carbon dioxide gas into the rectum or bladder for the purpose of killing the worms *in situ*.

Symmers,[2] in a paper describing a remarkable case, mentions that he has twice found living worms in the pulmonary blood, and describes a peculiar condition of polypoid outgrowths on the serous coat of the ileum, cæcum and colon, extreme polyposis of the large bowel, a fibrosis of the appendix vermiformis, the presence of eggs in the pancreas and lymphatic glands, and the typical liver cirrhosis, although there was only incipient bilharziosis of the urinary bladder.

Williamson,[3] in a paper on the disease in Cyprus, shows how it was connected with bathing in a certain river, while a suggestive article on Endemic Hæmaturia in South Africa, by Stock,[4] draws attention to the presence of fat in the urine and mentions the "toxin" treatment advocated by Birt. He cites two cases which contracted enteric fever and, as a result, were apparently cured of their bilharziosis, and a case of fatal dysentery in a native where, within two hours of the patient's death, the worms, on being dissected out, were found to be dead. He suggests repeated small doses of Wright's anti-typhoid serum, and mentions two cases under this treatment, of which, however, I can find no further record.

Letulle[5] has a paper on intestinal bilharziosis, and mentions the occurrence of the disease due to *S. mansoni* in the lesser Antilles, particularly in Martinique. He specially points out that it is solely confined to the lower end of the intestinal tract.

The paper is well illustrated and discusses the morbid histology of the lesions very fully.

Manson[6] adopts Sambon's classification, already mentioned, and regards *S. mansoni*, which has lateral-spined ova, as being probably a West African species which has been introduced into the western hemisphere by the African negro. It was first found by him in a West Indian patient whose urine was free and who had never suffered from hæmaturia. Sambon[7] has recently again dealt with this subject, and points out that he based his differentiation of the two species on differences in the structure of the female genital tract and on the ova, which are distinguished not only by the position of the spine but by its size and shape and by their own anatomical differences. He also considers the peculiar geographical distribution and anatomical habitat of *S. mansoni* as proof of its being a new parasite. A concise account is given by Manson of *S. japonicum*, the trematode found by Katsurada in human stools and in the portal system of cats in Japan, and discovered independently by Catto in a Chinaman's meso-colon in Singapore. Manson's book is, however, in every practitioner's hands and need not be quoted here. The occurrence of this parasite in the Philippine islands has been noted by Woolley.[8] He found lesions in the lung, liver and bowel of a Filipino and noted fibrosis of the liver.

Logan[9] describes three cases in China and gives rough drawings of the eggs and free embryos as they appear in the fæces. He thinks the fact that the egg is only a little larger

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[1] Sandwith, *Practitioner*, October, 1904.

[2] Symmers, W. St. C., "Studies in Pathology." Aberdeen, 1906.

[3] Williamson, G. A. (November 9th, 1907), "A Further Note on Bilharzia (Schistosomum) Disease in Cyprus." *Journal of Tropical Medicine*, p. 1333.

[4] Stock, P. G. (Sept. 29th, 1906), "Endemic Hæmaturia." *Lancet*, p. 857, Vol. II.

[5] Letulle, M. (April 15th, 1905), "Intestinal Bilharziosis." *Archives de Parasitologie*, p. 329, Vol. IX.

[6] Manson, Sir Patrick, "Tropical Diseases." 4th Edition, 1907.

[7] Sambon, L. W. (January 11th, 1908), "The part played by Metazoan Parasites in Tropical Pathology." *Lancet*, p. 102; and (January 15th, 1908). *Journal of Tropical Medicine and Hygiene*, p. 27, Vol. XI.

[8] Woolley, P. G. (January, 1906), "The Occurrence of Schistosomum Japonicum vel Cattoi in the Philippine Islands." *Philippine Journal of Science*, p. 83.

[9] Logan, O. T. (February 16th, 1906). "Three cases of infection with Schistosomum Japonicum in Chinese subjects." *Journal of Tropical Medicine and Hygiene*, p. 294.

than that of *Ascaris lumbricoides* is very important for the novice in fæcal examinations to note.

Dr. Low's[1] note on making permanent preparations of bilharzia eggs may be quoted. The little shreds of mucus passed with the urine are mounted in glycerin jelly and the cover slips ringed with canada balsam or asphalt.

It is perhaps worth mentioning here that there is a *Schistosomum bovis* of cattle and sheep, first described by Sonsino in Egypt, while Montgomery[2] describes a new species *S. indicum*, affecting horses and donkeys in India, and has also found two new species, *S. bomfordi* and *S. spindalis*, in Indian cattle. He also records the fact that a very large number of human cases were introduced into India from South Africa, and that the former country is evidently well-suited to the propagation of the Bilharzia parasite.

That bilharziosis occurs and is endemic in the Sudan has been shown in the First and Second Reports of these Laboratories. Only *S. hæmatobium* has, so far, been found. That it is also frequently being introduced from Egypt there can be no doubt, and as irrigation schemes increase, so will, in all probability, the amount of bilharziosis. At present the infection is probably limited, as regards its source, to the Nile, though, if the view be correct that the embryo reaches its human host in the body of some crustacean, then well-water may also be implicated. Time has not permitted further experiments with the species of Ostracode mentioned in the Second Report, but certainly the results obtained were suggestive. It is difficult to know if anything could be done to check the probable increase of this disease. At present it is not much in evidence, save amongst those who have lived in Egypt, and, strictly speaking, it would be well to guard the Sudan against it in somewhat the same manner as has been suggested for ankylostomiasis. Practically, however, any such scheme would, under existing conditions, almost seem impossible of realisation, though, if it could be properly carried out, the urine of immigrants likely to be bilharzia-carriers systematically examined, and those found infected refused admittance to the country or, at least, placed under medical control, I believe a possible danger might be averted. Bilharziosis is a serious menace to health in South Africa and fills the hospitals in Egypt. Hence it would be well to limit it as much as possible in the Sudan, and a sanitary policy directed to this end, though it may be regarded as Utopian, has much to commend it, while if it is to be introduced at all, the present is the time for action.

Blackwater Fever. The precise nature of this dreaded complaint, and one which has taken toll of several valuable lives in the Sudan, still remains unsolved. The chief views regarding it are:—

1. It is due to quinine acting under certain conditions and usually on a person the subject of malaria. This view is quite untenable, as is clearly shown by Manson.[3] At the same time, quinine can and does produce hæmoglobinuria. This is one of the rarer toxic effects of the drug.

2. It is a manifestation of malaria, either a severe form of the disease or a symptom of the concurrence of a kidney lesion with malaria, a view strongly urged by Plehn. Buchanan[4] has pointed out that three factors may be operative—malaria, quinine and the kidney lesion.

3. That it is a specific disease due to a special blood parasite, in all probability one of the piroplasmata, which may be conveyed from the sick to the sound by means of ticks.

Having seen very little blackwater fever, one has no opinion to offer, but an observation by a layman who has had great experience of the disease, and has lost many friends and companions by reason of it, may not be without interest. He informs me that at least half the cases which came under his notice had recently suffered from acute gonorrhea. This may have resulted merely in a lowering of general vitality, rendering the patients more liable to serious disease; and, of course, it is certainly not operative in many cases of blackwater fever, but it may possess some interest in view of the theory which regards a kidney lesion

[1] Low, G. C. (February 16th, 1907). "Method of mounting specimens of Bilharzia eggs, embryos, etc." *Journal of Tropical Medicine*, p. 67, Vol. V.

[2] Montgomery, R. E. (January and February, 1906), "Observations on Bilharziosis among Animals in India." *Journal of Tropical Veterinary Science*, p. 15, Vol. I.

[3] Manson, Sir Patrick (1907), "Tropical Diseases." 4th Edition.

[4] Buchanan, W. J. (April 27th, 1907), "The Third Factor in the Etiology of Blackwater Fever." *British Medical Journal*, p. 990, Vol. I.

Blackwater Fever— *continued*

as one of the essential factors. I merely mention it here as I can find no reference to its having been noted in connection with blackwater fever.

Christophers and Bentley[1] observed a phagocytosis of red blood corpuscles in the spleen of a case of blackwater fever. They specially note that the engulphed erythrocytes contained no parasites, so that the condition is different from what is seen in canine piroplasmosis where the phagocytosed red cells always contain piroplasmata. They strongly incline to the view that blackwater fever is the result of malarial infection. As regards the significance of the condition they describe, they think that if the phagocytosis of apparently normal red cells be taken in conjunction with the generally recognised fact that exposure for a certain time to malarious conditions is necessary before blackwater fever can be contracted, then it must be admitted that under certain conditions at some stage in the process of malarial immunisation, a process which is known in some degree to occur, there results a liberation of specific poison from the red cells, causing the extensive destruction of these elements which is the essential feature of the disease.

To medical officers in the Sudan, notes on new or recent methods of treatment are likely to be more serviceable than a recounting of various etiological theories.

Védy,[2*] a French doctor with much experience, believes the disease to be due to a toxin probably elaborated by a special micro-organism. His routine treatment consists of free purgation followed by frequent enemata, and in serious cases saline infusion. These measures are for the elimination of the supposed toxin, and are supplemented by the administration of warm water and weak tea by the mouth. Symptoms are treated as they arise; tendency to heart-failure, by caffeine and champagne; vomiting after the first day, by morphine and counter-irritation. The use of antipyretics and digitalis is contra-indicated, while pilocarpine is stated to be dangerous in this disease. This author also gives useful rules as regards the giving or withholding of quinine.

1. If, twenty-four hours after the onset, malaria parasites are present in the blood, give a small dose (12 grains) of quinine.

2. Never give quinine if malaria parasites are not present in the blood.

3. If in doubt (if an examination of the blood is not practicable), do not give quinine.

Hearsey's method, which is a modification of that of Sternberg for yellow fever, consists in the administration of 10 grains of sodium bicarbonate and 30 minims of the liquor hydrargyri perchloridi. The mixture is given every two hours for the first twenty-four hours and thereafter every three hours until the urine is free from hæmoglobin. Hearsey[3] recorded 18 consecutive cases treated in this way without a single death. The accompanying treatment consisted of milk and barley water given frequently and in small quantities. Champagne and acid drinks are eschewed, brandy being the stimulant employed when required. Benger's food is stated to be of great value. During convalescence the scaly preparations of iron were found most suitable as blood tonics.

Boxer[4] lays great stress on proper nursing and rectal feeding. He condemns the exhibition of quinine and thinks all drugs are better avoided, except perhaps calomel given as a purgative.

Owing to its anti-hæmolytic action, Vincent[5] recommended the administration of chloride of calcium in doses of 4 to 6 grammes by the mouth, or 1 to 2 grammes subcutaneously dissolved in physiological salt solution.

Hartigan[6] suggests, but it is merely a suggestion, the use of euquinine, the ethyl-carbonate, owing to its being a non-irritant, while Cook, quoted by Harford,[7] describes the practice in vogue in the German colonies, where cases are not invalided home, but if they

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[1] Christophers, S. R., and Bentley, C. A. (March, 1908), "Note on the Phagocytosis of Red Blood Corpuscles in the Spleen of a Case of Blackwater Fever. *Indian Medical Gazette*, Vol. XLIII., No. 3.

[2] Védy, L., "La fièvre bilieuse hæmoglobinurique dans le basin du Congo." Paris, A. Maloine, 1907.

[3] Hearsey, H. (March 5th, 1904), "The Treatment of Hæmoglobinuric Fever." *British Medical Journal*, p. 544, Vol. I.

[4] Boxer, E. A. (May 7th, 1904), "Hæmoglobinuric Fever." *British Medical Journal*, p. 1078, Vol. I.

[5] Vincent, H., *C. R. Soc. Biol.*, t. LIX., 1905, pp. 633, 635.

[6] Hartigan, W. (January 15th, 1907), "Euquinine—Its Suggested Use in Blackwater Fever." *Journal of Tropical Medicine and Hygiene*, p. 17, Vol. X.

[7] *Arch. f. Schiffs. u. Trop. Hyg.*, January, 1906.

* Article not consulted in the original.

Blackwater Fever—*continued*

have survived a first attack are put on gradually increasing doses of quinine each day, the urine being examined for the presence of hæmoglobin. If this does not occur in it, and if there be no rise in temperature, jaundice, or liver pain, the dose is run up to 15 grains, and this is then given every 8th or 9th day, with, it is said, the result that neither malaria nor blackwater fever occur. Cook himself employs Hearsey's treatment with apparently good results.

Mayer[1] described an interesting case which was treated by four-hourly saline enemata day and night—one pint being given at a time, during the height of the fever. Quinine was added to some of the latter enemata. The patient made a good recovery and the author notes that he was remarkably comfortable, that there was no vomiting during the whole course of the illness, that there was a regular rise of temperature every evening probably due to his being supplied with fluid, and that this rise yielded to treatment with small doses of quinine freely diluted. It should be noted, however, that the nursing was apparently ample and good.

Skelton[2] (Sierra Leone) distinguishes between hæmoglobinuric paludism (true blackwater) and quinine intoxication. He gives quinine by rectal injection, first of all administering a soap and water enema. The medicinal enema consists of quinine sulphate 5 grains, dilute hydrochloric acid *q.s.* to dissolve the quinine, and warm water 3 ounces. He gives opium for vomiting, 1/3 grain morphia repeated, if necessary, in 6 hours. As soon as the stomach will retain it he gives quinine by the mouth.

Dammermann[3]* reports favourably on the use of the decoction of the leaves of an African plant, *Combretus raimbanthius*, together with milk and potassium acetate. He gives his decoction in a strength of 24 parts to 1500 of water as a prophylactic to persons in whom quinine is apt to induce blackwater.

A practical point is mentioned by Mense,[4]* who finds that the kidneys are well flushed by large quantities of warm tea, best sucked through a tube, as this method tends to prevent vomiting. For this symptom Gush[5] recommends an effervescing mixture of carbonate of ammonia, sodium bicarbonate and citric acid. I have examined several blood films from blackwater cases occurring in the Bahr-El-Ghazal and have never found parasites of any kind present. In one case, which terminated fatally in Khartoum and has been recorded by Crispin,[6] I found the urine, which was at the time free from hæmoglobin, loaded with uric acid. This case was from the Blue Nile, but the patient had previously suffered from the disease in Central Africa. Recently, a primary case has occurred at Roseires on the Blue Nile, a place with an evil reputation for malaria. One cannot, however, be quite certain if this was a true blackwater case or a severe case of malaria in which hæmoglobinuria occurred.

Blood. Under this heading no allusion will be made to blood parasites. It is intended to deal very briefly with questions of morphology, clinical technique and medico-legal examinations which may furnish useful information to workers in the Sudan and other tropical countries.

Hankin[7] describes methods for the recognition of blood and seminal stains, especially in tropical climates. He points out that the high temperature of tropical climates has a two-fold action on blood and seminal stains. If the latter are kept damp they are apt to putrefy, if dry they become so insoluble as to be acted on with difficulty by ordinary reagents. In a blood stain so altered he finds that the absorption bands of hæmochromogen can be obtained, even when the blood-colouring matter is in an apparently undissolved and insoluble condition, by the following method :—

If on clothing, cut the stain out and plunge into boiling water for a few moments. Then place on a glass slide and wet with ammonium sulphide. Examine under the microscope

[1] Mayer, T. F. G. (December 2nd, 1907), "A Case of Blackwater Fever, Treated by Saline Enemata." *Journal of Tropical Medicine*, p. 378, Vol. X.

[2] Skelton, D. S. (June, 1908), "Some Observations on Blackwater Fever." *Journal of the Royal Army Medical Corps.*

[3] Dammermann, *Deutsche Med. Wochen*, 1906, No. 23.

[4] Mense, *Arch. f. Schiffs. u. Trop. Hyg.*, January, 1906.

[5] Gush, H. W. (December 16th, 1907), "Prophylactic and Remedial Treatment of Blackwater Fever." *Journal of Tropical Medicine and Hygiene*, p. 401, Vol. X.

[6] Crispin, E. S. (August 5th, 1905), "A Case of Blackwater Fever." *Lancet*, p. 357, Vol. II.

[7] Hankin, E. H. (November 10th, 1906), "Methods for the Recognition of Blood and Seminal Stains Especially in Tropical Climates." *British Medical Journal*, pp. 1261, 1843, Vol. II.

* Article not consulted in the original.

and move the specimen until the whole field of view is occupied by a portion of the coloured material. If necessary an oil immersion must be used. Remove the eye-piece and replace by a micro-spectroscope. If the stain is of blood the two absorption bands of hæmochromogen will be seen. If invisible, as a result probably of commencing putrefaction, a drop of a 10 per cent. solution of potassium cyanide should be allowed to fall on the stain, and the bands will appear somewhat nearer the red end of the spectrum than usual. The boiling is to prevent the colouring matter going into solution and being so diluted that the bands could not be seen. Stains on weapons or jewellery should first be wetted with ammonium sulphide. A small portion may then be scraped off with a knife and treated as above.

A new method of employing the guaiac test had been introduced by Holland[1]* owing to the difficulty of getting really old turpentine or good peroxide of hydrogen. He employs as an oxidising agent, sodium perborate, made from sodium dioxide and boric acid. Freshly broken pieces of guaiac resin are dissolved by boiling with alcohol in a test-tube for a few minutes till the tincture is yellow. The suspected material is then cautiously mixed with a drop or two of the guaiac solution to make a milky mixture. This is brought in contact with a fragment of sodium perborate on a white plate.

If the proportion of blood be large, the white perborate turns blue in a few minutes and remains blue until the drying of the guaiac leaves a yellow residue which changes the blue to green. If small, the white perborate turns a pale blue which becomes green as the guaiac dries. The test is simple and delicate, but is, of course, liable to the fallacies belonging to the ordinary guaiac reaction.

Turning to clinical methods, we find that Leishman[2] describes a simple method of enumerating leucocytes.

Two pipettes are employed—one, an ordinary one-cubic centimetre pipette graduated in 1/100ths of a cubic centimetre; the other, a capillary pipette to deliver five cubic centimetres. This quantity of the blood to be tested is taken up in the capillary pipette and at once diluted 200 times by being blown out into a watch-glass containing 995 cubic millimetres of water. Hæmolysis occurs but the leucocytes remain unaltered. Stir, shake and, after the capillary pipette has been washed and dried in the flame, take up with it two successive volumes of five cubic millimetres each and discharge them side by side as small drops on a clean slide. Allow these to dry and stain with Leishman's stain.

Count all the leucocytes in each drop with the help of a ruled cover-glass, prepared by allowing a drop of Leishman's stain to evaporate on the well-polished surface of the glass and ruling on the thin film which is left a series of parallel lines with the point of a sharp needle. A drop of cedar oil is placed on the stained drop film and the cover glass dropped on it, ruled surface downwards. Count, with a 2/3rd inch lens, the leucocytes in all the drops, representing the 10 cubic millimetres of the diluted blood. Multiply by 20 and you get the number per cubic millimetre of undiluted blood. The error, compared with a Gower's hæmocytometer count, seems to be about minus 5 per cent., which may be allowed for or neglected.

General clinical methods of enumerating leucocytes, including new and simplified procedures, are described by Turton,[3] but lack of space forbids a review of his paper.

Of more interest to the ordinary blood examiner are certain papers on Hæmiconia, what used to be called blood dust. Love[4] draws attention to the special prevalence of hæmiconia in typhus fever and describes four forms of bodies. 1. Protoplasmic bodies with bright refractile spots whose origin presents no difficulty, as staining shows them to be derived from fragmented neutrophile cells. 2. Small, round, highly refractile bodies from 0·5 to 1μ in diameter, and apparently motile. 3. Rod-like bodies, also apparently motile, from 0·5 to 2μ in length. 4. Dumb-bell forms, from 2 to 4μ in length, and apparently motile. He regards the last three as of the same class and mentions their incessant dancing movements and the fact that they cannot be stained. From this, and from their disappearance during the fixing process, he concludes that they cannot be derived from the disintegration or fragmentation of leucocytes or red blood corpuscles.

Porter[5] describes five forms. *a*. Greyish-blue, flagellated bodies of indefinite shape and possessing a twisting or rotatory movement. *b*. Bodies like a large diplococcus with rapid

[1] Holland, J. W. (June 8th, 1907). *Journal of American Medical Association*. Chicago.

[2] Leishman, W. B. (March 31st, 1906), "A Simple Method of enumerating Leucocytes." *Lancet*, p. 905.

[3] Turton, E. (February 25th, 1905), "Clinical Methods of Enumerating Leucocytes." *British Medical Journal*, p. 410.

[4] Love, A. (December 29th, 1904), "Hæmiconia." *Lancet*, p. 1781.

[5] Porter, F. (December 21st, 1907), "Observations on Blood Films, with Special Reference to the Presence of Hæmiconia." *British Medical Journal*, p. 1773.

* Article not consulted in the original.

movement. *c.* Bodies of indefinite shape, dumb-bells, rods, knobbed at one end, like a tadpole, etc., with slow movements. *d.* Small, round, vesicular, highly refractile bodies with a central ruby-coloured spot. These are usually quiescent, but may move slowly. *e.* Small, very rapidly moving, highly refractile micrococcal forms. Attempts at staining and cultivation failed. Porter believes that some are escaped nuclei of leucocytes, some escaped granules of leucocytes, others portions of disintegrated red cells, and that all are produced by some change in the blood constituents. **Blood—** *continued*

Nuttall and Graham-Smith[1] describe very similar forms, and state that they are liable to be mistaken for free forms of piroplasmata. In a later paper[2] they describe and figure the curious changes red corpuscles undergo in blood films, bodies being produced which might deceive the very elect. No one who has done much blood work but has encountered and probably been puzzled and deceived by some of these bodies. In my own experience the small, colourless, spherical forms have proved most troublesome, especially when working with fowl's blood. They are probably the free granules of leucocytes, but it is curious that they cannot be stained :—

One of the most useful and practical papers which has recently appeared is that by Sutherland on "The Differential Diagnosis of Tropical Fevers." It occurs as an appendix to Chapter III. of Roberts'[3] admirable work on Enteric Fever in India. Here we need only note some of the remarks on leucocytes :—

"A leucocytosis or relative increase of the lymphocytes or of the polymorphonuclears in the circulation, with absence of parasites in the peripheral blood, spleen or lymph, is always suggestive. A lymphocytosis points to tuberculosis or to a bowel infection by one of the typhoid or allied groups, and a polymorphonuclear cytosis to a local septic infection. A lymphocyte increase is of less value in diagnosing local infections than an increase of the polymorphonuclears, and calls for the diazo reaction, the agglutination and sedimentation tests and the search for tubercles in the choroid with the ophthalmoscope to clear up the issues. Increase of the polymorphonuclears, on the other hand, is distinctive, for it means local septic infection somewhere."

A long list of what has to be looked for follows, in which one specially notes oral sepsis, sore throat, appendicitis and liver abscess.

One may add to these notes as the result of the work of Stitt, Vedder, Ashburn and Craig, and to a less extent from personal observation, that a decrease in the polymorphonuclears and a marked increase in the small lymphocytes points to dengue fever, especially if there is an accompanying leucopenia. This will be considered later. A useful paper on the conditions producing eosinophilia is that by Fearnsides,[4] who in a summary states that the condition is usually associated with the presence of *Schistosomum hæmatobium*, *Trichinella spiralis*, *Ankylostoma duodenale*, the various species of *Filariæ* and *Echinococcus cysts*. It may also occur associated with the presence of any one of the *Helminthidæ*, but is rare in cases infected with *Dibothriocephalus latus*, and not common in infections with *Trichocephalus trichiurus*. He further points out that the changes in the leucocytes are to be regarded as due to toxic agents produced by the worms, and in the nature of a reaction for the good of the host.

Emery,[5] in his useful clinical work, gives an easy method of recording the differential leucocyte count, which does not seem to be very generally known and certainly saves much time. "The simplest way of noting down the leucocytes," he says, "is to assign letters to each variety, P for polynuclear, E for eosinophile, etc., and to put these down in blocks of five each, thus :—

P P P L E
P P L L H
L P P P P
P L L P L
P P P L P"

In this way you can tell at any time how many leucocytes you have counted. I should think that anyone who has made differential counts in the heat of the Sudan by the ordinary method of headings and columns will appreciate this simplified and rational procedure.

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[1] Nuttall, G. H. F., and Graham-Smith, G. S. (October, 1906), "Canine Piroplasmosis." *Journal of Hygiene*, p. 586.

[2] Nuttall, G. H. F., and Graham-Smith, G. S. (April, 1907), "Canine Piroplasmosis." *Journal of Hygiene*, p. 586.

[3] Roberts, E., "Enteric Fever in India, etc., etc." London, 1906.

[4] Fearnsides, E. G. (March, 1906), "The Effects of Metazoan Parasites on their Hosts." *Journal of Economic Biology*, p. 41, Vol. I.

[5] Emery, W., "Clinical Bacteriology and Hæmatology." 2nd Ed. London, 1906.

Blood— *continued*

Rogers[1] states that the count can be much shortened and simplified by enumerating only 250 leucocytes. This is done by counting backwards and forwards from edge to edge of the best part of the blood film, avoiding the thick end and the "tag" end. He only counts polynuclears, large mononuclears including transitional forms, lymphocytes and eosinophiles. He uses a 1/8-inch or Zeiss D objective and considers as large mononuclears only such mononuclear cells as are as large as, or larger than, an average polynuclear, the smaller ones being classed as lymphocytes. By this method he obtains reliable results. While for rough and ready work for clinical purposes this is no doubt a very useful and rapid method, I[2] have pointed out that in accurate estimations the leucocyte classification adopted by Dutton and Todd seems to be the best, and that it is necessary to have some generally employed classification for comparative purposes.

The error of a hæmocytometer count and the method of correcting the same is discussed by Student[3*] and noted in the epitome of the *British Medical Journal*.[4] It need not be discussed here but the reference may be found useful.

Horrocks and Howell[5] describe and illustrate some curious X-bodies which they found in Spain in the blood of patients suffering from an ill-defined form of fever and in cattle which were not healthy.

As in the blood of a sick dog in Khartoum, examined by Mr. Archibald, I have seen bodies exactly like some of these described, and, as the condition may yet prove to be an important one, I quote their description of the bodies stained by Leishman's method:—

> The bodies, when stained, were characterised by a faint capsule with a circular centre staining deep blue; they varied in size, some being as large as a red corpuscle, others only about one-eighth the size of a red corpuscle. In addition to these forms, which were the most common, the following were also seen:
>
> (*a*) A small, blue circular centre surrounded by four or more faint capsules concentrically arranged; (*b*) two circular bodies, each having a dark blue central point surrounded by a light blue ring, enveloped in one capsule which appeared indented as if two capsules were in process of formation; (*c*) similar to (*b*), but the part surrounding the deep blue centre stained a deeper blue, and two indented capsules were seen; (*d*) a dark blue central part, shaped like a crescent, containing a small circular body, with a deep blue central point within the arms of the crescent. None of the bodies on the slide showed any signs of chromatin.
>
> Intravenous inoculation of a rabbit gave positive results.

The authors were unable to pronounce on the precise nature of these bodies, which, however, proved not to be acid fats. In fresh blood the bodies showed no amœboid movement.

Bubo. A case of climatic bubo in Uganda is described by Castellani.[6] Blood and bacteriological examinations were negative. He mentions that the disease occurs chiefly on the east coast of Africa, the West Indies and Straits of Malacca and China. It has not been recorded from Central Africa.

Cantlie and Hewlett[7] discuss the relation of climatic bubo to plague. Cantlie named it pestis minor, although pus from the affected glands proved sterile. Cantlie and Hewlett record a case where three bacteria were grown from the excised gland, *i.e. Staphylococcus pyogenes albus*, *Staphylococcus cereus albus*, and a minute bacillus staining by Gram's method and curdling milk. The last-named was non-pathogenic to guinea pigs and mice, and corresponded to a micro-organism isolated by Kitasato from a case of plague. Simpson stated that climatic bubo seemed to bridge over the true plague epidemics. Wright looked upon the disease as distinct from plague, while Emery regarded the organism in question as possibly the acne bacillus of *Sabouraud*, which might have reached the glands from the skin. Clayton[8] reports four cases in which he performed blood examinations, finding in two of

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[1] Rogers, L., "Fevers in the Tropics." London, 1908.

[2] Balfour, A. (April 1st, 1907), "Notes on the Differential Leucocyte Count, with Special Reference to Dengue Fever." *Journal of Tropical Medicine*, p. 113.

[3] Student, "*Biometrika*," Vol. V., part III., pp. 351–360.

[4] *British Medical Journal*, p. 154, Vol. II., January 18th, 1908, "The Error of a Hæmocytometer."

[5] Horrocks, W. H., and Howell, H. A. L. (April, 1908), "X-bodies found in the Blood of Human Beings and Animals." *Journal of the Royal Army Medical Corps*, Vol. X., No. 4.

[6] Castellani, A. (December 15th, 1903), "Climatic Bubo in Uganda." *Journal of Tropical Medicine*, p. 379.

[7] Cantlie, I., and Hewlett, R. F. (April 4th, 1904), "Bacteriology of Climatic Bubo." *British Medical Journal*, p. 593, Vol. I.

[8] Clayton, T. H. A. (January 2nd, 1905), "Notes on Climatic Bubo." *Journal of Tropical Medicine*, p. 1, Vol. VIII.

* Article not consulted in the original.

them marked eosinophilia, which he suggests as due to toxin action. He comes to no definite conclusion regarding the cause of the disease but quotes several arguments against the pestis minor theory. **Bubo—** *continued*

I do not know if climatic bubo has been observed in the Sudan. It is possible that it occurs in the southern districts contiguous to Uganda, but I have never heard of its being reported.

Cachexial Fever. *See* Leishmaniosis *(page* 95*)*.

Calabar Swellings. The relation of this condition to *Filaria loa* and *diurna* is dealt with in the *Journal of Tropical Medicine*, 1/7/04. Amongst other places these are found on the Upper Congo, so that it is quite possible they may occur in the Bahr-El-Ghazal Province. Manson thinks it practically certain that they are somehow produced by *F. loa*, though the mechanism of their production is unknown. Their sudden appearance, gradual disappearance, painlessness, and the fact that they never suppurate, sufficiently distinguish them. The only human filaria I have found in the Sudan is *F. perstans*, and it occurred in a Ugandese.

Cancer. The literature on cancer during the past few years has become enormous, and one can only direct attention to a few points, such as the possible parasitic origin of the disease, supposed preventive methods, and its occurrence in the coloured races and in tropical countries.

Ford Robertson and Wade[1] described bodies like the *Plasmodiophora brassica* which is known to cause tumours in cruciferous plants. These are only demonstrable by special metallic processes. In a later paper[2] they describe the technique and also methods of culture which they maintain were successful, and discuss the probable etiological relationship of these parasites to carcinoma. The tendency of other observers was to regard these bodies as cell inclusions. Ford Robertson and Young,[3] however, in a still more recent article, deal with cyanide-fast bodies in tumour cells and describe improvements in the technique of preparing and staining tissues by their special processes. They also note a great activity of polymorphonuclear leucocytes which they believe to be directed against a specific parasite. Still more recently the senior author[3] describes rod-shaped bodies, something like tubercle bacilli but evidently not bacteria, in certain carcinomata. He believes these to be a stage in the life-history of the protozöon found by himself and Wade, and that several allied species are concerned in tumour production.

Interest for a time centred round the *Micrococcus neoformans* of Doyen, but the most recent work, including that of Dudgeon and Dunkley,[4] discredit it as a cause of cancer. These authors have shown that it is an organism of very low pathogenicity, and that the serum of patients suffering from malignant disease does not develop any very marked agglutinative property for *M. neoformans*. In fact, it is less than that which is found for the *Staphylococcus albus*.

Mention should be made of the work of Gaylord and Calkins[5] who found a special spirochæte, *S. microgyrata* (Lowenthal), in primary and transplanted carcinoma of the breast of mice. It does not stain by Giemsa.

Two papers which have at least the merit of being interesting and practical are those of Keetley[6] and of Brand.[7] The former is strongly in favour of the parasitic theory, and lays down very stringent prophylactic rules which at the present day would be difficult to enforce in their entirety amongst all classes, however desirable they may be. He says:

1. Sterilise the food, and points out that it is where food tends to tarry that cancer of the alimentary tract is apt to develop. 2. Ensure a sufficient and regular toilet and protection of the nipples and genitalia.

[1] Robertson, F., and Wade, H. (August 13th, 1904), "Cancer and Plasmodiophoræ." *Lancet*, p. 469, Vol. II.

[2] Robertson, F., and Wade, H. (January 28th, 1905), "Researches into the Etiology of Carcinoma, etc." *Lancet*, p. 215, Vol. I.

[3] Robertson, F., and Young, C. W. (August 10th, 1907), "Researches into the Etiology of Carcinoma; Notes upon the Features of Carcinomatous Tumours revealed by an Improved Ammonia-silver process." *Lancet*, p. 359.

[4] Dudgeon, L. S., and Dunkley, E. V. (January, 1907), "The Micrococcus Neoformans." *Journal of Hygiene*, p. 13, Vol. VIII.

[5] Gaylord, H. R., and Calkins, G. N. (April 10th, 1907), "A Spirochæte in Primary and Transplanted Carcinoma of the Breast in Mice." *Journal of Infectious Diseases*, p. 155, Vol. IV.

[6] Keetley, C. B. (October 13th, 1906), "The Prevention of Cancer regarded as a Practical Question Ripe for Solution." *Lancet*, p. 993, Vol. II.

[7] Brand, A. T. (January 11th, 1908), "Some Remarks on the Infectivity of Cancer." *Lancet*, p. 80.

3. Take care of mouth and teeth. 4. Destroy dressings from discharging, malignant ulcerations. 5. Attend to non-malignant sores and tumours. Excise cancerous and doubtful tumours early. 7. Practise abstinence from alcohol, tobacco, excessive meat eating, and foods which leave waste products. 8. Avoid all unnecessary familiarity, especially with strangers. 9. Attend carefully to kitchen hygiene and the hygiene of food generally.

Brand advances many very suggestive points, especially on the infectiousness and auto-inoculability of cancer, and points out that it is impossible for the "carcinoma cell" to be the true parasite as suggested by Butlin. He recommends the examination of fresh, living carcinoma cells on the warm stage of the microscope, and suggests that the new device of Gordon which enables a good magnification of 7000 diameters to be obtained, and the system of dark-field illumination introduced by Siedentopf may greatly facilitate cancer research. He strongly advocates cleanliness in its widest sense, showing how very readily food, especially vegetables and fruit, can become contaminated, and denounces earth burial, advocating cremation.

As regards distribution, Sutherland[1] presents statistics for the Punjab, which, as he says, tend to show that cancer is not a common disease there, but that such cases as occur apparently affect all classes. He also notes that the nature of the diet does not seem to affect the incidence of cancer in the Punjab. The same, he says, is true of alcohol, syphilis and malaria. From India[2]* during 1904, 146 cases of malignant new growth were reported to the Imperial Cancer Research Fund amongst vegetarian natives, 137 amongst natives living mainly on flesh diet, and 222 amongst natives living on a mixed diet. "Cancer in the British Colonies" is the title of a paper in the *Journal of Tropical Medicine* of March 1st, 1905,[3]* and a point of interest to us in the Sudan is the statement that the disease has not been seen amongst natives of the Gambia, Ashanti and Natal, and that it is said to be rare in British Central Africa, the Eastern African Protectorate, Southern Nigeria and on the Gold Coast.

Thus Hearsey says that amongst the natives of British Central Africa, though cancer occurs, it is of the utmost rarity, while non-malignant growths are relatively common.

Amongst the Chinese, cancer is rare (Clark), and the same is true of the Malay States, Jamaica and Ceylon, while in British New Guinea, where be it noted the Papuans cook all their food and live chiefly on vegetables and fish, the disease seems to be absent (Craigen).

As regards the Sudan, I have records of only ten cases of malignant tumour examined in these laboratories during the past five years. Of these, half were carcinomatous and half sarcomatous. I think it may be taken that malignant disease is not common amongst natives of the Sudan even though in the northern parts the native, in many places, has come into association with Europeans. I cannot say much about the matter from a clinical standpoint, but I understand that most cases of new growth dealt with surgically at the Military and Civil hospitals in Khartoum are sent to the laboratories for diagnosis and, if this be the case, neoplasms play no great part in the pathological field.

Dr. Waterfield of the Sudan Medical Department confirms this statement, and his experience goes to show that tumours of all kinds are rare in the Sudan.

At my request, Colonel Hunter, P.M.O., kindly sent out a letter of enquiry to his Medical Officers, asking for their opinions regarding the prevalence of cancer in their districts.

Captain Thompson, writing from Wad Medani in the Blue Nile Province, reports:—

"So far as I have been through this Province up to date, I have seen no cases of malignant disease." He adds: "I may say that I saw a case of scirrhus of the breast in a woman at Kassala in 1906, and a suspicious case in a boy in this Province, who, however, did not come here for treatment as directed."

Curiously enough, shortly after this was written Captain Thompson sent in for examination the tissues from a well-marked case of epithelioma of the foot.

Captain Brakenridge, S.M.O., Bahr-El-Ghazal, answered:—

"I have never seen a single case of malignant disease in this Province in about twenty-one months I have served here. In fact, in my eight years' service in the Egyptian Army I only remember to have seen one epithelioma of the tongue in a Sudanese, and one sarcoma of the neck in an Egyptian."

Captain Anderson replied from El Obeid, Kordofan:—

"During a year spent in the Province, in which time I have inspected widely in all directions and seen many hundred sick, I have never come across a single case of malignant disease amongst the Arab, Nuba and

[1] Sutherland, D. W. (November, 1904), "Cancer in the Punjab." *Indian Medical Gazette*, p. 425, Vol. XXXIX.

[2] "Scientific Reports of the Imperial Cancer Research Fund," No. 2, Part I, 1905.

[3] Quoting "Blue Book on Cancer in the Colonies, 1906."

* Article not consulted in the original.

Misad tribes. This is almost as remarkable as the virtual non-existence of tubercular disease in Kordofan. Microbic diseases (thanks, I imagine, to the wide air space and strong sun) are universally rare, while parasitic complaints—malaria, guinea worm, tænia and other intestinal worms—are of common occurrence." **Cancer—** *continued*

Captain Bousfield, of Kassala, stated that :—

"During my year's stay in this Province I have not seen a single case that I could definitely diagnose as malignant disease, either amongst the civil or the military. In the Military Hospital, Kassala, there have been no cases of malignant disease during the years 1904–1907 inclusive. At Gedaref there has been a case, scirrhus of the breast in a woman aged about 45, and a doubtful case of sarcoma of the leg (possibly a mycetoma) in a man aged about 40. There have been no cases that could certainly be diagnosed as malignant disease in the Kassala Civil Hospital. My own opinion is that epithelioma, carcinoma and sarcoma are extremely rare in this Province."

It is worthy of note that all these regions are somewhat remote, and the natives inhabiting them do not come much into touch with Europeans. With the exception of the Bahr-El-Ghazal, which is a negroid district, the prevailing type of inhabitants is the Arab, who, however, very frequently has much negro or other blood in his veins.

Cerebro-Spinal Fever. If the subject of cancer has no very intimate relationship with the Sudan, the reverse is true of cerebro-spinal fever, which, in former years, was much in evidence so far as can be ascertained, and accounted for many deaths, while every now and again sporadic cases or small epidemics occur under existing conditions. It was Buchanan who drew special attention to its appearance during the dusty months of the year in India, and doubtless the inhalation of dust plays a part in its propagation.

Goodwin[1]* in a large number of cases found the *meningococcus* present in the nasal cavity, while Vansteenberghe and Grysez[2] discovered it in the noses of healthy men, cultivated it, and proved that the cultures, when injected under the meninges of rabbits or guinea pigs, produced the typical lesions of cerebro-spinal meningitis. The necessity of dealing with infected nasal discharges is therefore apparent and disinfection of the nasal cavities of the patient and all contacts is clearly indicated.

The same points are also brought forward by Fraser and Comrie,[3] who record that hot, dusty, ill-ventilated atmospheres, which provide conditions favourable to the growth of the *meningococcus* and to the occurrence of naso-pharyngeal catarrh, are often associated with the dissemination of epidemic cerebro-spinal meningitis. They also state that "the high comparative percentage of fathers whose naso-pharynx was found by us to contain the *meningococcus*, points to the fact that they probably are the carriers of the disease to their children."

Speer[4]* describes an early pressure symptom which he has found regularly present together with Kernig's sign. It consists of a turning in of one or both feet until, if not disturbed, one lies across the other. The legs later become flexed and tend to cross each other. The symptoms, he states, are due to a combination of toxin poisoning, nerve irritation and pressure. The *Indian Medical Gazette* for September, 1905, publishes the leaflet issued by the German Health Department. The only points requiring notice are that children attending school, although in good health, must be kept from school if they live in the same house as the patient, until the medical authorities permit their re-admission to school, and the recommendation of a weak solution of menthol for the disinfection of the throat and hands. Under the heading "Nursing" one notes that the gargling water of the patient should be disinfected. As regards the rash, Chalmers[5] describes a case in a child where the features of a typhus rash were exactly reproduced, while Wright[6] gives an account of the rashes met with in the Glasgow epidemic. These were usually hæmorrhagic and of a purpuric nature, varying in size from a mere point to something smaller than a lentil, and often being grouped in clusters. Occasionally these spots came out in crops. In one case he describes purple and maroon spots together with larger, pale-blue blotches on the trunk and limbs.

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[1] Goodwin (November 11th, 1905). *Medical Record.*

[2] Vansteenberghe, P., and Grysez (January, 1905), "Contribution à l'Etude du Meningocoque." *Annals de l'Institut Pasteur*, p. 69, t. XX.

[3] Fraser, J. S., and Comrie, J. D. (July, 1907). *Scottish Medical and Surgical Journal.*

[4] Speer, G. G. (May 15th, 1905). *Medical Record.*

[5] Chalmers, A. K. (July 7th, 1906), "The Rash of Cerebro-Spinal Fever." *British Medical Journal*, p. 23.

[6] Wright, W. (September 15th, 1906), "The Rash in Cerebro-Spinal Meningitis." *Lancet*, p. 717, Vol. II.

* Article not consulted in the original.

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l Fever
ntinued

Steven,[1] in an interesting lecture, deals with the differential diagnosis, mentions a case where fæcal poisoning was mistaken for a case of cerebro-spinal meningitis, and refers to an Egyptian case of verminous infection closely simulating the fever.

Nedwill[2] records two epidemics in the Sudan occurring in the summer of 1905 and 1906, during the months, be it noted, when the dust storms occur. From a study of 22 cases he concludes that non-recovery of the knee jerks within a week of the onset of the disease is an unfavourable sign. The mortality was roughly 59 per cent.

Cases of remarkable cure after the use of collargol are reported.[3] It is employed as an injection into the spinal canal, doses mentioned being 0·05 gramme and 5 c.c. of a 1 per cent. solution.

The claims of Weichselbaum's *meningococcus* to be the cause of the disease have been amply confirmed by recent work to which there is no need to allude, but mention may be made of the bacillary form which this organism may assume when cultivated. A note on this will be found in a paper by Darling and Wilson[4] who from their work conclude that the *Meningeal diplococcus* belongs to the *Streptococcus fæcalis* group, and is identical with the *Micrococcus rheumaticus*.

The latter author[5] in a later paper states that all the Gram-negative cocci met with by him and his co-workers in cases of cerebro-spinal meningitis failed to grow on the Drigalski-Conradi medium. To this rule he found three exceptions, and the diplococci from these cases not only grew well on this medium, but, instead of tending to take on a bacillary form as is usual, retained their diplococcal characters on the Drigalski-Conradi medium, although they tended to assume the bacterial form on agar. "We conclude then," he says, "that in the lumbar puncture fluid of certain cases of cerebro-spinal meningitis Gram-negative diplococci may be found which differ from Weichselbaum's and Still's cocci in respect of their morphology and capacity for growth on the Drigalski-Conradi medium. It may be that certain abnormal appearances presented by meningococci, such as growth in short chains which competent observers claim to have seen, may have been due to the presence of this coccus in the cultures."

The opsonic power of the serum has been the subject of research by various workers, and Houston and Rankine[6] tabulate the results of their examinations, finding that the opsonic index seems to be a more delicate test of infection than the agglutination reaction, and that the two tests combined will prove of great value in diagnosis. Levy[7]* records a remarkable series of cures by means of Kolle-Wassermann's serum injected intra-spinally. The dose for children over one year was 20 c.c., for adults 30 to 40 c.c. Of 23 cases treated with the serum, only 5 died, and of these 3 had too small a dose or received the dose subcutaneously. Of 17 cases properly treated, only 2 died. A preliminary injection of morphine is given, then the serum, and the patients are kept for eight to twelve hours with their pelves raised. Robb[8]* speaks favourably of Flexner and Jobling's serum, but does not commit himself to a definite opinion as to its merits. Trautmann and Fromme[9]* record the results of work done in the Hygienic Institute in Hamburg during 1907. Thirty-two specimens were examined from patients and 312 from contacts. In only 9·2 per cent. of the latter was Weichselbaum's meningococcus isolated. One "germ-carrier" case remained infective for 66 days. In subcultures on Lœffler's serum a typical growth was common,

[1] Steven, J. L. (September 8th, 1906), "Epidemic Cerebro-Spinal Fever, with Illustrative Cases." *Lancet*, p. 638, Vol. II.

[2] Nedwill, C. L. (December 1st, 1906), "Cerebro-Spinal Meningitis in the Sudan." *Lancet*, p. 1502, Vol. II.

[3] January 12th, 1907, "Recovery from Cerebro-Spinal Meningitis under Injections of Collargol in the Spinal Canal." *Lancet*, p. 106, Vol. I.

[4] Darling, J. S., and Wilson, W. J. (February 23rd, 1907), "A Case of Cerebro-Spinal Meningitis." *British Medical Journal*, p. 433, Vol. I.

[5] Wilson, W. J. (June 20th, 1908), "Differentiation of certain Gram-negative Cocci occurring in Cases of Cerebro-Spinal Meningitis by their Morphology and Power of Growth on the Drigalski-Conradi Medium." *Lancet*, Vol. I.

[6] Houston, T., and Rankine, J. C. (May 4th, 1907), "A Note on the Opsonic Power of the Serum, with Reference to the Meningococcus of Cerebro-Spinal Fever occurring in the Belfast Epidemic." *Lancet*, p. 1213, Vol. I.

[7] Levy, G. *Deutsche Med. Wochen.*, 1908, No. 4, p. 139.

[8] Robb, A. G. (February 15th, 1908), "The Treatment of Epidemic Cerebro-Spinal Fever by Intra-spinal Injections of Flexner and Jobling's Anti-meningitis Serum." *British Medical Journal*, p. 382.

[9] Trautmann, H., and Fromme, W. (1908). *Münch. Med. Wochenschr.*, No. 15.

* Article not consulted in the original.

Cerebro-Spinal Fever —*continued*

in primary cultures involution forms often occurred. The organism was found to ferment glucose and maltose, but not levulose. The diagnostic value of the agglutination test is slight. The *Diplococcus crassus* which occurs along with the meningococcus is agglutinated by meningococcus serum. A valuable and very practical paper is that by Robertson[1] of Leith, who deals specially with administrative control, and advocates the douching of the nasal cavities of all "intermediaries" with chlorine water. His method was to douche at intervals of two days, and three times in all. He also draws attention to the value of formamint lozenges, especially for children. He also recommends the isolation of all those living in infected houses. Thorough spraying of infected premises with formaldehyde was deemed useful, and, a point which might be missed, the confiscation and destruction of all foodstuffs found in lower class houses is stated to be a valuable preventive measure.

As regards diagnosis, Birnie and Smith[2]* successfully isolated and cultivated the specific organism from the blood by the simple procedure of puncturing a vein and distributing 4 cubic centimetres of blood equally between two flasks containing 75 cubic centimetres of sterile bouillon. Kutscher[3] finds an agar, made with human placental juice, an excellent medium for the growth of the first generation of the *meningococcus*.

In the only case I have seen in the Sudan I was able to isolate and cultivate a *diplococcus* from the meninges, which answered in every respect to that of Weichselbaum.

Chlorine water would probably be of little use as a nasal douche in this country, but the menthol wash recommended by the Germans might be tried. It would, I think, be comparatively easy, in the light of recent knowledge, to control an outbreak in Khartoum, where the people are amenable to sanitary control, and very thorough disinfection methods followed by compensation can often be adopted owing to the small value of native dwellings and belongings. [*Note.*—A recent outbreak has enabled one to prove the truth of this assertion.]

Chicken-pox. In the Sudan, where one deals chiefly with black skins, the diagnosis of chicken-pox from small-pox is sometimes very difficult. The following points, which have served one as fairly trustworthy guides, and have been gathered from various sources, may be helpful. Rogers[4] suggests that the blood changes in the two conditions might well repay study:—

1. Prodromata. Often no prodromal period in chicken-pox. Usually present in small-pox.
2. Feeling of illness when rash appears in chicken-pox. The opposite is true in mild or modified small-pox.
3. Facial appearance. Nothing special in chicken-pox; heavy, anxious or stuporose in small-pox.

Amongst the natives these three are of less value than the following:—

4. Frequently a rise in temperature accompanies appearance of rash in chicken-pox. In small-pox the temperature falls at this time.
5. Rash appears first on the trunk in chicken-pox, on the face in small-pox.
6. Distribution of rash. Trunk and proximal portions of extremities in chicken-pox. Face and distal portions in small-pox, together with back of trunk. (*See*, however, note under "Small-pox," *page* 183).
7. If a so-called "skin window" be marked off, the irregularity of the rash is well seen in chicken-pox. *i.e.* vesicles and pustules together in the area. Not so in small-pox.
8. Rapid change from papule to vesicle in chicken-pox, frequently in a few hours and within 24 hours. At least 24 hours in small-pox, often 72 hours.
9. Centre of vesicle its highest point in chicken-pox; depressed in small-pox.
10. Papules of chicken-pox not so firm and shotty as those of small-pox.
11. Depth of skin involved. Less in chicken-pox than in small-pox. Hence "seeds" in palms and soles usually found only in the latter.
12. The character of the scales, thin in chicken-pox, thick in small-pox, is said to aid one, but I have not noticed this in native cases. Early cupped scabs in chicken-pox are, however, very characteristic.
13. The scars of chicken-pox are smooth and have irregular edges, while those of small-pox are pitted and as if punched out. The former are often wider as the vesicles tend to spread laterally.

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[1] Robertson, W. (July 27th, 1907), "Remarks on the Outbreak of Epidemic Cerebro-Spinal Meningitis." *British Medical Journal*, p. 185.

[2] Birnie, J. M., and Smith, M. T. (October, 1907). *American Journal of Medical Science.*

[3] Kutscher, K. (November 9th, 1907), "Ein Beitrag zur Züchtung des Meningococcus." *Cent. für Bakt.* Abt., 1907, Vol. XLV., No. 3, p. 286.

[4] Rogers, L., "Fevers in the Tropics," London, 1908.

* Article not consulted in the original.

n-pox Neech,[1] Rolleston[2]* and Porter[3] have recorded cases in which the eruption became
ntinued confluent, in this and other respects closely resembling that of small-pox. Mackenzie[4] thinks that varicella, "with its polymorphic eruptions, mature and immature developments and retrogressions, recurrent invasions, uneven temperature and irregular periods of incubation," must be regarded as a mixed infection, and that it is possibly "a non-specific, non-variolous varicella and a very slight but genuine *variola infantum* of childhood." Rolleston[5] has a paper on the accidental rashes of varicella, which in order of frequency are classed as scarlatiniform, purpuric, morbilliform and mixed. He mentions that there may possibly be a chicken-pox without vesiculation, and discusses the nature of the accidental rashes which are probably septic or toxic.

Bray[6] describes a condition amongst the Sudanese. It is called by them *Boorglum*, and is apt to be mistaken for chicken-pox. It is said to be most common at flood Nile, and takes the form of a superficial rash, papular, vesicular and pustular, affecting the back of the hands and forearms, the dorsum of the feet and the front of the leg. It is probably parasitic, is commonest in those who work with mud bricks, and is best treated by the application of iodine.

I have seen *Boorglum* in a Greek bricklayer, and the rash is certainly like that of chicken-pox. There is, however, no constitutional disturbance and the distribution of the eruption is different.

Chigger. As *Sarcopsylla penetrans* is well known in the Bahr-El-Ghazal, and has caused much invaliding amongst men in the Sudanese battalions, the following points in its life-history elicited by Wellman[7] are likely to prove useful:—

1. The eggs are always laid while the chigger is yet embedded in the flesh of her host. Her different behaviour after artificial removal does not form a real exception to these statements. In such an event she extrudes all her eggs at once and dies, but such eggs do not hatch into larvæ. Even if the chigger has completed her gestation and has begun to lay her eggs before her removal, only the most mature eggs in the posterior part of the abdomen will develop.

2. They never (at least in this climate, Angola) hatch into larvæ in the body of the parent.

3. They are not laid at one time in masses, but discreetly, and sometimes at considerable intervals, depending on circumstances. If the chiggers are in the sole of the foot, and the infected person walk about, the eggs may be seen dropping from his feet as he goes, or by pressing gently on the skin near a ripe chigger two or three eggs may be seen to escape one after another. Such eggs readily hatch out. So while it is doubtless a good rule to burn all chiggers removed, yet this has no effect on the usual mode of propagation, and so long as natives go about with infected feet the cycle will go on.

4. The shell of the parent when dead, and empty of eggs, usually dries up *in situ* and causes no further trouble. Occasionally it may cause irritation, swellings and ulcers, but most of the abscesses, sores, etc., from chiggers come from removing the insects with septic instruments.

5. The mature ova, if placed in a glass-covered dish containing some dust from the floor, go through the developmental stages common to all fleas, which have often been described. In natural conditions they develop in the dirt and cracks in the floor, and in chigger countries it is therefore important to compel one's native servants to keep their feet clear of chiggers, and to allow no other natives (especially children, who are always infested) in one's quarters.

Cholera. The Sudan has always been liable to invasion by cholera from Egypt and from the great pilgrimage centres on the eastern coast of the Red Sea. Given invasion, the disease is now more likely to be disseminated owing to improved methods of communication and especially to the establishment of the Atbara-Port Sudan Railway. Hence any acts relating to cholera must ever be of interest to the Medical Officer, and more especially when these deal with preventive measures. The useful Indian pamphlet by Duke[8] served as a basis for the cholera notices drawn up this year for Khartoum City and Khartoum North, and to be issued if the disease became epidemic in any part of the country. As these are possibly of some general interest they are here introduced although, happily, their utility or otherwise has not so far had to be tested.

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[1] Neech, J. T. (February 24th, 1906), "A Note on a Case of Confluent Varicella." *Lancet*, p. 515.

[2] Rolleston, J. D. (January, 1906). *British Journal of Children's Diseases.*

[3] Porter, C. R. (May 18th, 1907), "A Case of Confluent Hæmorrhagic Eruption in Varicella." *Lancet*, p. 1352.

[4] Mackenzie, J. (January, 1907), "A Study in Varicella." *Journal of Royal Institute of Public Health*, p. 17.

[5] Rolleston, J. D. (May 4th, 1907), "Accidental Rash of Varicella." *British Medical Journal*, p. 1051.

[6] Bray, H. A. (October, 1904). *Journal of the Royal Army Medical Corps.* London.

[7] Wellman, F. C. (December 1st, 1905), "A Point in the Life-History of Sarcopsylla Penetrans." *Journal of Tropical Medicine*, p. 394.

[8] Duke, J., "The Prevention of Cholera, and its Treatment." 3rd Edition. Calcutta, 1905.

* Article not consulted in the original.

Cholera is in the majority of cases a water-borne disease, due to water having become contaminated with the cholera organism derived from some person suffering from the disease. **Cholera—** *continued*

It is, therefore, usually conveyed by the drinking of water which has become polluted by the excreta or discharges of an infected person, as such water may occur in the form of ice, or may be found added to milk, or used for the washing of vegetables, etc.; ice, raw milk and uncooked vegetables, salads and fruit may also transmit infection. Flies also and other insects, especially ants, may be to blame by carrying the infection from polluted matter to food and drink, while these may be contaminated by the infected and dirty hands of those engaged in their preparation. The organism is easily killed by boiling and drying, hence the value of the following:—

PREVENTIVE MEASURES

(i.) All water intended for personal use, viz., drinking, cooking, washing, and, wherever possible, bathing, must be boiled. Drinking water is best boiled in a can provided with a cover and a tap. The boiled and cooled water can then be drawn directly into the cup or tumbler. Care must be taken that an infected drinking vessel is not used, as, after the boiled water has cooled, it can be re-infected. Water from zeers and goulahs should be looked upon with suspicion unless these are carefully watched and cleaned.

(ii.) All raw milk must be boiled.

(iii.) Uncooked vegetables, raw salads and fresh fruits, especially melons, should be avoided.

(iv.) Food stuffs should be carefully protected from flies, ants and other insects, and not stored anywhere in the proximity of latrines or any collection of refuse. As far as possible all food should be cooked. Jellies are liable to become contaminated and should be avoided.

(v.) Personal cleanliness on the part of those engaged in preparing food and drink is most essential. Care should be taken to see that cooks and other servants are cleanly in their habits and clothing, and are careful to wash their hands before handling food or dishes or vessels used for food or drink.

(vi.) General cleanliness, especially in latrines and in kitchens and cook-houses is most necessary. All dishes should be carefully cleaned with boiling water, and kitchen cloths should be well washed and dried in the sun. Any cloths used for straining soups, sauces and the like should be washed in permanganate solution *(vide infra.)*. Brooms, brushes, or cloths used for cleaning out latrines must on no account be used in kitchens or cook-houses.

(vii.) In any case of cholera or disease like cholera, with diarrhœa, colic, vomiting or cramps in the arms, legs or stomach, the vomit or stool should be kept covered up until seen by a Medical Officer. The latter should at once be informed of the illness, and only those in actual attendance on the patient should be permitted to stay in the room with him.

Great care must be taken thoroughly to wash and disinfect the hands immediately after touching the patient or the bed-clothes or any vessel containing his vomit or dejecta.

The same care is necessary on the part of those dressing or burying the corpse of anyone dead of the disease.

(viii.) Any symptoms like those occurring early in cholera, especially colic and diarrhœa, should at once be treated. In order to enforce these precautions, and for the general information of the public, it is hereby notified:

1. That no water will be allowed to be taken from the river bank. Anyone attempting to do so will be liable to imprisonment or punishment.

2. That no bathing or washing will be permitted from the river bank, nor will anyone be permitted to foul the bank, or the river from the bank, in any way whatever under pain of punishment.

3. Pure water may be obtained from such wells in the town as have been disinfected, and inhabitants are warned against using water from any untreated well. Bathing and washing in the immediate vicinity of wells is prohibited.

It is hoped that a general supply of pure water will be distributed both in Khartoum and Khartoum North.

4. The general public milk supply will be placed under sanitary control, and inhabitants are hereby warned to obtain their milk only from one or other of the Municipal Milk Depots. The site and arrangements of these will be duly notified later.

5. All ærated water factories will be placed under sanitary control, and only such ærated waters as can be drunk with safety will be issued.

6. The ice factory will be placed under sanitary control, and only such ice as can be used with safety will be issued.

7. Disinfectants will be issued at cost price from the office of the Sanitary Inspector in the Mudiria. Purchasers must bring their own bottles. Instructions for the disinfection of wells, latrines and kitchen floors will be issued separately.

8. The use of weak tea and lemon drinks made with boiling water is hereby recommended, as is an early application for preventive medicines in all cases of colic or diarrhœa.

9. The inhabitants are informed that cholera is a complaint which is very easily prevented and controlled provided the necessary measures are taken, and they are invited to co-operate with the authorities and to assist them to cope with the disease.

Ants as carriers of infection were specially included, because in Khartoum they are more in evidence than flies, and I am certain that from their crawling habits and scavenger propensities they can play a considerable rôle in the infection of food and drinks.

olera—
continued

From a paper by O'Gorman[1] one picks out the following practical points as likely to be of service. Note the stage when called to a case, as the treatment varies with the stages of the illness. Look for a blood-shot condition in the eyes, sometimes the only outward indication of reaction. Take the temperature in the axilla in preference to the mouth or rectum (dangerous and unnecessary). Remember the differential diagnosis from arsenical poisoning. The author recommends as a routine practice in every case and at any stage the exhibition of calomel and sodium bicarbonate in doses of 3 to 6 grains and 6 to 12 grains respectively, repeated if rejected, until retained. The calomel increases the flow of bile, acts as an intestinal antiseptic, is sedative to vomiting, especially in frequently repeated fractional doses, gr. 1/10 to gr. 1/20, every quarter or half-hour, is diuretic, antiphlogistic, and, taken continuously in doses short of toxic, stimulates the faculties, physical and mental. Soda aids its action, prevents salivation and supplies a vital element to the blood.

In the first stage he also recommends the administration of intestinal antiseptics such as sulpho-carbolate of zinc, copper arsenite, acetozone, medical izal or medical cyllin.

For the rest, stop food, give fluids and try to prevent collapse. Carminatives, sedatives and astringents are useful and should be given. He mentions chlorodyne, camphor and opium amongst the drugs, and states that nuclein may prove very valuable owing to the increase of polynuclears it produces and its stimulation of cell growth.

In the stage of collapse he utters a warning against alcohol, and states that there are only two great remedies, namely, atropine and strychnine. He regards these as sheet anchors in cholera.

In the case of both drugs small doses frequently repeated are best, and the strychnine should be pushed and any ill effects neutralised by chloral. The value of heat, sinapisms and warm rectal injections is mentioned. In the stage of reaction, he points out that the occurrence of urination is a favourable sign and where there is danger of uræmia, pilocarpine may be tried. It is, however, risky, and diaphoretics and hot coffee are safer and often efficient.

The article concludes with advice as to diet, sanatogen, somatose and plasmon being mentioned. This treatment certainly does not err in the way of doing too little, and possibly the writer is over-fond of medicaments and expects too much from them, but he has evidently had large experience of cholera and gives definite and apparently sensible reasons for his recommendations.

It is curious he does not mention the red iodide of mercury treatment in extreme collapse, so strongly advocated by Duke (with whom he is otherwise more or less in agreement), who gives dilute sulphuric acid in the early stage together with cannabis indica, and for suppression of urine recommends subnitrate of bismuth and turpentine with cupping to the loins, and, if required, pilocarpine controlled by strychnine.

Both mention the necessity of an early aperient dose in some cases, the one recommending sulphate of magnesia, the other castor oil with tincture of belladonna.

Choksy[2] has a paper advocating cyanide of mercury in doses of 1/10th of a grain every two or three hours as a germicidal agent. He reports favourably on its use, the only drawback being a tendency to stomatitis during convalescence. In other directions his treatment is like that of O'Gorman. In acute delirium during the reaction stage bromide and hyoscyamus are indicated.

McCombie[3] reports very favourable results from employing subcutaneous injections of salt solution (60 grains to a pint of boiled water) at a temperature of from 115° F. to 110° F., repeated whenever the pulse tended to fail. The addition of adrenalin chloride (1 in 1000) to the pint of salt solution also seemed beneficial.

Rogers and Mackelvie[4] speak highly of the value of large quantities of hypertonic salt solutions in transfusion for cholera. The strength they employ is just about two drachms to the pint, and they inject, as a rule, four pints at a time, intravenously. Subcutaneous injections are only of benefit in mild cases. By this new procedure they believe the

[1] O'Gorman, P. W. (November, 1905), "How to Cure Cholera." *Indian Medical Gazette*, p. 414, Vol. XL.

[2] Choksy, Khan Bahadur, N. H. (April 20th, 1907), "Some Indications for the Treatment of Cholera." *Lancet*, p. 1077.

[3] McCombie, F. C. (May 26th, 1906), "A Note on the Treatment of Cholera by Saline Injections." *Lancet*, p. 1468.

[4] Rogers, L., and Mackelvie, M. (May, 1908), "Note on the Value of Large Quantities of Hypertonic Salt Solutions in Transfusion for Cholera." *Indian Medical Gazette.*

mortality has been halved. They have also found repeated dry cupping over the loins of great service in uræmic cases. **Cholera—** *continued*

Waters[1] praises izal. He made stock solutions, of which each fluid drachm contained fifteen minims of izal made up with tragacanth mucilage. Each drachm was diluted with seven of water, and this dose of 1 oz. was given every hour or two hours as long as necessary. He had no less than 41 recoveries out of 56 cases.

Banerji[2] testifies to the value of the eucalyptus treatment introduced by Major Harold Browne. The oil was given in 5 minim doses, together with mucilage and syrup of lemons. Thirty-three cases, most of them in the stage of collapse, were treated and the percentage of recovery was sixty-three. The oil is said to act both as an antiseptic and stimulant.

Other points of interest are the "latent" cholera carriers found by Gotschlich[3] at Tor, who, though they harboured true cholera vibrios, did not give rise to an epidemic and did not die of cholera, but from dysentery and gangrene of the bowel; and the quick agglutination method of diagnosis introduced by Dunbar[4*] and said to be reliable. It is as follows:—

Mix a particle of fæcal mucus with 1 drop of peptone water and 1 drop of a 1 in 500 dilution of cholera serum (mixture *a*). Mix a similar particle with 1 drop of peptone water and 1 drop of a 1 in 50 dilution of normal rabbit's serum (mixture *b*). Place each mixture on a cover glass and examine as a hanging drop preparation. Observe agglutination in *a*, none in *b*. Maximum result after about 3 hours. In this connection one must cite the work of Ruffer,[5] which leads him to state that "it is not advisable to trust to the agglutination test only in bacteriological diagnosis of cholera. The test is useful but not specific." It would seem then that the hæmolysis test must always be applied, for Ruffer noted no vibrio hæmolyses, when the agglutination test, Pfeiffer's reaction and the fixation test are positive, while he states distinctly that "the agglutination, saturation and Pfeiffer's tests are not in themselves of absolute diagnostic value for cholera vibrios."

Some of Ruffer's results have been called in question, and it is very desirable that his conclusions should be definitely confirmed or confuted, as they upset prevailing ideas on the bacteriological diagnosis of cholera.

Recent work on anti-cholera serum, Strong's new prophylactic, etc., though important and suggestive, scarcely comes within the scope of this *résumé*.

Climate. Sandwith[6] has a paper on hill stations and other health resorts in the British Tropics. He deals with Egypt and then goes on to speak of the Sudan, which he says:—

> Is less destitute of mountains, and the Government has now established a sanatorium at Erkowit (3500 feet), 30 miles due west of Suakin and 22 miles due east of Summit Station on the Nile Red Sea Railway, from which there is a motor road. The best season is from May to September, when the headquarters of the Suakin province move there. It is now utilised for change of air by officials from Suakin, Port Sudan and Khartoum, and has the great advantage of being uninhabited by natives. Visitors now live in tents, but temporary rest houses are being built.
>
> Unlike the neighbouring hills, which consist of bare ironstone and diorite, there is here wonderful vegetation, grass, maiden-hair fern, many shady trees and fine open spaces large enough for playing polo and golf. There are easy walks to neighbouring hill crests, from which magnificent views of the Red Sea coast can be obtained. The climate is relatively cold, very fresh and invigorating for all convalescents. Vegetable gardens have been planted and some Southdown sheep have been imported by the Governor of the province but most food has to be conveyed from Suakin. There is a good supply of water from a spring in the hills, and soundings are now being taken to find other sources. In order to keep the ground as clean as possible native tribes with their camels, cattle and goats, are forbidden to enter the station.

He has a few notes on Sinkat, also, and concludes thus: "There is no special sanatorium for the Blue and White Nile or for the Bahr-El-Ghazal, which is the most tropical part of the Anglo-Egyptian Sudan." It is found by experience better to allow convalescents to spend a fortnight in Khartoum before they proceed to Cairo and Europe.

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[1] Waters, E. E. (December 1905), "The Treatment of Cholera with Izal." *Indian Medical Gazette*, p. 451.

[2] Banerji, H. C. (January, 1905), "Oil of Eucalyptus in Cholera." *Indian Medical Gazette.*

[3] Gotschlich, F. (1906), "Uber Cholera und Choleraähnliche Vibrionen unter den aus Mekka Zurückkehrenden Pilgern." *Zeit. für Hyg. u. Inf. Krank.*, p. 281.

[4] Dunbar. *Berliner Klin. Wochen.*, 1905, No. 39, p. 1237.

[5] Ruffer, M. A. (March 30th, 1907), "The Bacteriological Diagnosis of Cholera." *British Medical Journal*, p. 735.

[6] Sandwith, F. M. (November 15th, 1907), "Hill Stations and other Health Resorts in the British Tropics." *Journal of Tropical Medicine and Hygiene*, p. 361, Vol. X.

* Article not consulted in the original.

Climate— *continued*

It is not quite the case that no natives inhabit Erkowit. They are accustomed to graze their animals at these altitudes which they visit periodically, and indeed there is a native cemetery on the ground. At certain seasons Erkowit is wrapped in damp mists, but on the whole the description given is correct, and, though not ideal, the station is likely to prove valuable.

In summing up, Sandwith points out the absurdity of sending a patient suffering from the dire effects of malaria to a health resort where he can become re-infected, and speaks of the necessity for maps showing the distribution of malaria-bearing mosquitoes. In their absence, he says, we must be content to judge by altitude, and, in a country where malaria is endemic, regard any height under 5000 feet as unsatisfactory.

In 1906 the larvæ of *Pyretophorus costalis* were brought me from a water-course at the base of the Erkowit plateau. I reported the matter and mentioned its importance, but so far as I know further action was not taken until I sent Mr. King specially to Erkowit for the purpose of determining precisely the species of mosquito present and their distribution. He did not find Anophelines at or near Erkowit, but discovered a new species of sand-fly and certain mosquitoes which are described in his report.

Sandwith points out that malarial patients are apt to get fever attacks when exposed to cold altitudes, sea breezes, or even the damp cold of countries such as England. He believes this to be largely due to an insufficient quinine treatment. Cantlie,[1] on the other hand, from personal experience, records his belief that "it is not the cold but the hot weather in Britain, especially in the south of England, that has to be dreaded by the old tropical resident who is the subject of chronic malaria. As far as the British Isles go, he recommends the climate of Morayshire and Nairn in the North of Scotland, while in the winter he believes in the Swiss mountains at an altitude of not less than 4000 feet.

Sandwith regards change to a temperate climate as essential in bad cases of sprue and beri-beri, while for cases of dysentery and enteric the sea coast is recommended, but here also insufficient or improper treatment previously may be the chief cause of a relapse. Such cases sent to the hills in India are apt to contract hill-diarrhœa.

Much interesting information regarding climate will be found in Woodruff's book,[2] which, however, has to be read as a whole and cannot well be quoted here. A good deal that he brings forward is not applicable to the Northern Sudan, for he deals chiefly with typical tropical conditions where heat and moisture are combined to the greater detriment of those exposed to them.

Wolfe[3]* has investigated the effects of climates on American soldiers stationed in the Philippines. His paper is of a preliminary nature, but he notes that the kind of life led by the individual has much to do with the change produced. A more or less active life is necessary. The more indolent the life the sooner the stagnation and retrogression. Men, however, break down under excessive marching in the Tropics quicker than in temperate climates. The points observed were: (1) The pulse rate taken during sitting, standing and exercise; the maximum and minimum rates of the pulse; (2) the respiration; (3) the hæmoglobin; (4) erythrocytes; (5) leucocytes; (6) differential leucocyte count.

A remarkable paper is that by MacDonald,[4] who, dealing chiefly with tropical Queensland, advocates an active out-door life, his motto apparently being "the more sun the better," and this for man, woman and child. He advances proof to show that under such conditions the white race thrives; this even in a country where the rainfall is heavy. His arguments certainly tend to upset all prevailing ideas on the subject, and do not seem to have been taken very seriously by those who discussed a paper which, whatever its value, is certainly interesting and perhaps suggestive.

Haldane[5] has discussed the influence of high air temperature and has conducted experimental work on the subject. He quotes the old experiments carried out by Blagden,

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[1] Cantlie, J. (June 15th, 1907), "Clinical Observations on Tropical Ailments as they are met with in Britain." *British Medical Journal*, p. 1455.

[2] Woodruff, C. E., "The Effects of Tropical Light on White Men." Rebman, London, 1905.

[3] Wolfe, E. P. (August 31st, 1907), "A Preliminary Report of Research Work on the Effects of Tropical Climate on the White Race." *Medical Record.*

[4] MacDonald, T. F. (May 1st and 15th, 1908), "Tropical Lands and White Races." *Journal of Tropical Medicine and Hygiene*, Vol. XI., No. 10.

[5] Haldane, J. C. (October, 1905), "The Influence of High Air Temperature." *Journal of Hygiene*, p. 494, Vol. V.

* Article not consulted in the original.

Forsyth and Dobden, in 1775, who found that they could remain for a few minutes in a room at about 250° F. (121° C.) without serious inconvenience or marked rise of body temperature although beef-steaks exposed in the room at the same time and place could be cooked within 13 minutes. Needless to relate the air was dry. A few of his conclusions may be cited. (*a*) The rectal temperature did not show any abnormal increase during rest in still air until the temperature by the wet-bulb thermometer reached about 88° F. (31° C.) provided the subjects were stripped to the waist or clad in light flannel. (*b*) In moving air (with the wet-bulb still below the body temperature) a higher wet-bulb temperature could be borne without abnormal rise of rectal temperature. (*c*) The symptoms observed to accompany the rise of body temperature were—(1) a marked increase in the pulse-rate, accompanied by throbbing in the head; (2) dyspnœa, particularly on any exertion; and (3) a general feeling of exhaustion and discomfort. These he points out depend to some extent on other causes than the rise of body temperature as indicated by the rectal thermometer. **Climate—** *continued*

Tyler[1] has introduced a new scheme for correlating personal sensations as regards climatic conditions with the ordinary measurement recorded by meteorological instruments. His paper is very erudite, and we may merely note that he finds that, except for any dry conditions of the atmosphere, the readings of the wet-bulb thermometer indicate very closely the degree of discomfort experienced due to temperature and humidity, and that these readings, or what he calls his "*hyther*"[2] degrees, form the best available means for comparing climates. It is not possible here to discuss the question of the climate of the Sudan, for at least three distinct varieties exist; that of the Red Sea littoral, that of the dry, sandy northern regions, and that of the humid and rainy south. Doubtless also the desert climate is modified by proximity to rivers, by elevation as in Kordofan, by the presence of vegetation as in some parts of the Kassala province. Some allusion to the climate of Khartoum will be found under "Sanitary Notes," this being the only part of the country about which one can speak as a result of any prolonged experience, and even then five years is no great length of time considering how climatic conditions often run in cycles.

Clothing. This question as regards the Tropics is briefly discussed in Woodruff's book,[3] but with special reference to the Philippines. It is pointed out that the outer day clothing should be white, grey, or yellow, the colours which absorb heat least; but, as white clothing freely transmits the actinic rays which are dangerous to the nervous system of white men, and the light rays said to produce skin disease in blondes, the underclothing should be opaque and black or yellow in colour.

Sambon's paper[4] advocating the use of "Solaro" fabrics may be read with advantage, this cloth being a successful attempt to obtain the ideal fabric for the white man in the Tropics, *i.e.* one which will at the same time exclude the harmful, short or actinic rays and reflect the heat rays, thus avoiding complexity of garments and much unnecessary weight.

Duke, in his pamphlet on cholera, strongly condemns the persistent use of a thick belt or kummerbund. It acts like a poultice, weakens the abdominal organs and actually tends to increase the action of the bowels. This, of course, does not apply to its use at night, especially when sleeping out of doors where there is a risk of chill.

A very practical and up-to-date paper, which takes care to consider tropical conditions, is that by Chesney.[5] The proper clothing for women receives due notice. Absorbent materials for underwear are condemned, and light non-absorbent clothing, which of necessity has to be frequently washed, is recommended. The author notes that in the Tropics the wearing of a cholera belt is not now an article of faith, and acquiesces in the more modern ideas regarding its usefulness.

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[1] Tyler, W. F. (April 15th, 1907), "The Psycho-Physical aspect of Climate, with a Theory concerning Intensities of Sensation." *Journal of Tropical Medicine*, p. 130.

[2] "Hyther"—the joint effect of temperature and humidity on human sensation.

[3] Woodruff, "The Effects of Tropical Light on White Men." London, 1905.

[4] Sambon, L. W. (February 15th, 1907), "Tropical Clothing." *Journal of Tropical Medicine and Hygiene*," p. 67, Vol. X.

[5] Chesney, L. M. (July, 1908), "Hygienic Clothing and Disease." *Journal of the Royal Institute of Public Health*, Vol. XVI., No. 3.

Dengue. Carpenter and Sutton[1]* investigated the pathology of dengue fever in 1904. They failed to find any organisms in the blood of dengue cases, nor were they able to implicate any of the mosquitoes with which they worked. *Culex fatigans*, however, was not one of these. In throat swabs a small diplococcus was found, either free or in the epithelial cells. A leucopænia was found present from the first, and it is suggested that a diplococcus or delicate, bipolar staining bacillus like Pfeiffer's bacillus of influenza may be the cause, infection taking place by way of the respiratory tract.

A full report of an epidemic in Brisbane in 1905 will be found in the *Journal of Tropical Medicine* for December 15th, 1905. In some instances the incubation period seemed as short as 24 hours. The characteristic "breakbone" pains were not much in evidence. A very minute account of the symptoms is given. As rare complications, pneumonia, pleurisy, parotitis and orchitis are mentioned. Ulceration of the oral mucous membrane and the fauces, and gingivitis were noticed. Diarrhœa with mucous evacuations was not uncommon and dysuria occurred. As sequelæ, boils and carbuncles, an itchy vesicular eruption of the hands, muscular rheumatism, neuralgias, giddiness, nervous depression and loss of memory are recorded. Eye lesions were fortunately rare. It is pointed out that the initial symptoms of dengue closely resemble those of yellow fever. In the differential diagnosis from influenza, stress is laid on the rash, not, however, a constant symptom, and still more on the almost invariable absence of catarrhal symptoms of the respiratory tract and the extreme rarity of pulmonary complications. The few differential leucocyte counts made did not show the apparently characteristic changes to be detailed immediately. No evidence is adduced as to etiology.

Stitt[2] has a paper on the blood changes, and details what he considers the most characteristic blood findings as follows:—

1. Absence of a demonstrable protozöon.
2. Leucopænia.
3. Diminution of polymorphonuclears.
4. A striking variation in the percentage of other leucocytes at varying periods of the disease. At first a large increase in the small lymphocytes is observed, then the appearance of a greater proportion of large lymphocytes, and in the final stages (at the time of the terminal rash and during convalescence) a most striking increase in the mononuclears.

Stitt failed to find the so-called protozöon described by Graham,[3] of Beirut, but certain observations led him to believe that some species of culex is very probably the transmitter of the disease.

The following are the important conclusions of Ashburn and Craig[4] as a result of their work on a dengue epidemic occurring near Manila in the Philippines:—

1. No organism, either bacterium or protozöon, can be demonstrated in either fresh or stained specimens of blood with the microscope.
2. The red-blood count in dengue is normal.
3. There occur no characteristic morphological changes in the red or white corpuscles in this disease.
4. Dengue is characterised by a well marked leucopenia, the polymorphonuclears being decreased, as a rule, while there is a marked increase in the small lymphocytes.
5. No organism of etiological significance occurred in broth or citrated blood cultures.
6. The intravenous incubation of unfiltered dengue blood into healthy men is followed by a typical attack of dengue.
7. The intravenous inoculations of filtered dengue blood into healthy men is followed by a typical attack of the disease.
8. The cause of the disease is, therefore, probably ultra-microscopic in size.

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[1] Carpenter, D. N., and Sutton, R. S. (January 21st, 1905). *Journal of American Medical Association.*

[2] Stitt, E. R. (June, 1906), "A Study of the Blood in Dengue Fever, with Particular Reference to the Differential Count of the Leucocytes in the Diagnosis of the Disease." *Philippine Journal of Science*, p. 511, Vol. I.

[3] Graham, H. (July 1st, 1903), "'The Dengue,' a Study of its Pathology and Mode of Propagation." *Journal of Tropical Medicine*, p. 209, Vol. V.

[4] Ashburn, P. M., and Craig, C. F. (June 15th, 1907), "Experimental Investigations Regarding the Etiology of Dengue Fever." *Journal of Infectious Diseases*, p. 440, Vol. IV.

* Article not consulted in the original.

9. Dengue can be transmitted by the mosquito, *Culex fatigans*, and this is probably the most common method of its transmission. **Dengue—** *continued*

10. The period of incubation in experimental dengue averages three days and fourteen hours.

11. Certain individuals are absolutely immune to dengue, as proved by our experiments.

12. Dengue is not a contagious disease, but is infectious in the same manner as is yellow fever and the malarial fevers.

In another paper[1] they deal with diagnosis. In differentiating from yellow fever the slower pulse, jaundice and hæmatemesis occurring in that disease are helpful. The same would hold good in the Egyptian disease most resembling yellow fever, namely, infectious jaundice. As regards influenza, they point out the association of dengue with the presence of mosquitoes, while influenza occurs where they are absent and often in cold weather. They mention the catarrhal symptoms in the latter and lay stress on the leucopænia and lymphocytosis found co-existing in dengue. Early small-pox has to be differentiated, and sometimes an acute follicular tonsillitis simulates dengue. The prophylaxis resolves itself into protection against mosquitoes. In this paper are recorded the differential blood counts by Vedder who assisted in the work. The variation in the relative proportion of the large and small lymphocytes found by Stitt was not confirmed, but, as already stated, his other results were substantiated.

Ross[2] has recently advanced strong confirmatory evidence to show that the immunity of Port Said from dengue fever since 1906, while epidemics raged elsewhere in Egypt, was due to the abolition of *Culex fatigans* in that town. The same is true of Ismailia, which escaped during the epidemic of 1907.

One[3] has been able to make some personal observations regarding dengue in the Sudan, but only on a small scale. As the disease was very prevalent in Egypt and parts of the Sudan during the summer and autumn of 1906, one was in hopes of being able to carry out a study of the blood in dengue. Fortunately in one sense, unfortunately in another, though Port Sudan and Halfa were visited by epidemics, Khartoum, so far as can be told, wholly escaped. Not a single case of dengue fever was notified, and this, although it is more than likely that several persons suffering from dengue must have reached Khartoum by railway from the north, while I saw one case which arrived in Khartoum before convalescence was fully established, and while he was probably still in an infectious state.

Is it not possible, then, that the immunity which Khartoum has enjoyed is due to the comparative freedom of the town from mosquitoes, and especially from *Culex fatigans?* No species of mosquito was at all common in Khartoum during the months when dengue was prevalent in other parts of the Sudan and in Egypt. Thus, during June, 1906, there were in Khartoum 689 water collections which might have served as mosquito breeding places. Of these 17 were infected with larvæ or pupæ, being 2·47 per cent. The corresponding figures for Khartoum North were 125; 4; 3·2 per cent. During July the percentage infected in Khartoum was 4·35, in Khartoum North 3·2; August, Khartoum 7·22, Khartoum North 3·20; September, Khartoum 9·94, Khartoum North, 3·20; October, Khartoum 8·32, Khartoum North 4·76. The slight rise in August, September and October was due to heavy rainfall, but adult mosquitoes were not numerous.

The figures are only approximately correct, but they give a good idea of the state of the town. One does not wish to press the point too much, but the observation is interesting and suggestive so far as it goes.

I append a table of differential leucocyte counts made on blood films from cases of dengue and supposed dengue sent me by Dr. Crispin from Port Sudan. It is necessary to note that one has classed as lymphocytes, both true lymphocytes and lymphocytes with irregular nuclei, while under the term mononuclears, are included both large lymphocytes and large mononuclears in accordance with the very useful classification of Dutton and Todd.[4] Transitionals, however, have been placed separately.

My cases were few in number, and most of the bloods were taken only in the early stages of the fever. Moreover, in one or two cases, I do not know what the eventual diagnosis was.

[1] Ashburn, P. M., and Craig, C. F. (May, 1907), "Experimental Investigations regarding the Etiology of Dengue Fever." *Philippine Journal of Science*, p. 71, Vol. II.

[2] Ross, E. H. (July 1st, 1908), "The Prevention of Dengue Fever." *Annals of Tropical Medicine and Parasitology*, Series T. M., Vol. II., No. 3.

[3] Balfour, A. (April 1st, 1907), "Notes on the Differential Leucocyte Count, with Special Reference to Dengue Fever." *Journal of Tropical Medicine and Hygiene*, p. 113, Vol. X.

[4] Dutton, J. E., and Todd, J. L. (1903). *The Thompson, Yates and Johnson Laboratories' Report*, Vol. V., New Series, Part 2, Liverpool.

Dengue— *continued*

DENGUE FEVER

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9*
	? Day of Fever	Second Day of Fever	Second Day of Fever	Third Day of Fever	Third Day of Fever	First Day of Fever	? Day of Fever	? Day of Fever	Ninth Day of Fever
Eosinophiles ...	1·75	1·25	2·5	1·5	·75	1·75	12·5	2	6·25
Polymorphonuclears	*27·25*	60	*33·5*	*57·25*	*35·25*	*84·25*	*27·75*	43	*44·25*
Mononuclears ...	27	12	14·5	7·5	10·25	8	5	12·75	26
Lymphocytes ...	*43·5*	25	*48·5*	*33*	*53·25*	5·25	*52·25*	38·5	*22·25*
Transitionals ...	1·25	1·75	1	1·75	·25	·5	·75	3·75	1·25
Basophiles	·25	...	...	·25	·25	·25	·5	...	...
Myelocytes	...	...	...	...	...	...	1·25	...	...

* Terminal rash fading, temperature normal.—Case 9 was a European. I believe all the other bloods were those of natives; Egyptians, Arabs or Sudanese.

In every instance 400 cells were counted. Still, in the apparently undoubted dengue cases (Nos. 4, 5 and 7), the results appear to confirm those of Stitt, though, as mentioned in my original paper, it is very necessary that some kind of standard classification of leucocytes be adopted whereby differential counts by various observers in all parts of the world may be made strictly comparable.

A paper by Saigh,[1] on dengue in Port Sudan, states that the cases there occurred chiefly in houses infested by mosquitoes (species not stated), and that the fever reappeared when there was an increase of mosquitoes in the town. Further, all persons living in the hospital escaped infection, and the hospital was the only place free from mosquitoes.

Phillips[2] in his Egyptian cases used aspirin for relieving pain, and found calcium chloride useful in hæmorrhagic and urticarial cases.

Dhobie Itch. This does not appear to be very common, at least in the Northern Sudan. The climate is probably too dry to favour the growth and proliferation of the germs. In one case I found what I believed to be *Microsporon minutissimum*. Chrysophanic acid ointment proved efficient. Glacial acetic acid has been recommended, and for natives strong liniment of iodine is most serviceable.

Diarrhœa. This is always an important question in the tropics owing to its relation with dysentery and sprue, but the infantile form also merits attention. Recently there has been much work done on infantile diarrhœa.

Hewlett[3] states that the *Bacillus dysenteriæ* is probably the etiological factor in various forms of infantile and epidemic diarrhœa. He mentions that Miss Wollstein isolated this organism in all (39 out of 114) cases of infantile diarrhœa where blood and mucous were present.

An article in the *Lancet* for September 17th, 1904, in dealing with errors of diet as a cause of infantile diarrhœa, mentions the septic variety which may attack strong as well as weakly subjects, and in which nervous collapse may continue after the diarrhœa ceases to be a cause for anxiety. Nash,[4] while admitting that there may be some connection between sub-soil temperature and the advent of epidemic diarrhœa, regards contamination of food by infected dust, and especially infection-conveying flies, as the main cause of the disease. "The essentials," he says, "for putting a stop to the great waste of infant life every summer are

[1] Saigh, S. (November 15th, 1906), "Dengue in Port Sudan, Red Sea Province." *Journal of Tropical Medicine and Hygiene*, p. 348, Vol. IX.

[2] Phillips, L. (December 15th, 1906), "Dengue in Egypt." *Journal of Tropical Medicine and Hygiene*, p. 373, Vol. IX.

[3] Hewlett, R. T. (April, 1904), "Dysentery and Infantile Diarrhœa, the Etiology of." *Journal of State Medicine*, p. 229, Vol. XII.

[4] Nash, J. T. C. (September 24th, 1904), "Some Points in the Prevention of Epidemic Diarrhœa." *Lancet*, p. 892.

(1) Clean milk supplies; (2) Clean towns with well-organised system of sewage removal, dust collection and disposal, and street watering; (3) Clean homes where sufficient domestic hygiene prevails to permit an understanding of the importance of clean utensils for food, the covering over of food to protect from dust and flies, and personal habits of cleanliness; (4) Inhibition of fly life." **Diarrhœa—** *continued*

In a later paper[1]* he states that there is no one specific micro-organism of diarrhœa and that he is not convinced that breast-fed infants are really liable to epidemic diarrhœa. Thus, amongst 138 deaths of infants under one year of age, there were 68 deaths from diarrhœa in hand-fed infants and not a single death from diarrhœa amongst the 28 who had been entirely breast-fed.

Hewlett[2] agrees on most points with Nash, admits with him that there is no specific micro-organism, but again states that the *B. dysenteriæ* is the causative organism in a large proportion of the cases, other organisms, such as *B. coli*, *Proteous vulgaris*, *Streptococci*, *B. pyocyaneus*, and perhaps others, being operative in the remainder. He thinks that infection of the food takes place mainly in the homes.

Griffith[3]* notes that bacteria of the lactic acid-producing group clearly exert an inhibitive influence upon some of the milk bacilli which are specially dangerous to infants, and concludes that this explains the value of milk purposely soured by adding the lactic acid bacillus—for instance, buttermilk, which is useful in diarrhœa. He thinks that heat also plays a part in infantile diarrhœa by its depressant action on the nervous and vaso-motor systems and by its interference with the digestive processes.

Sandilands[4] considered epidemic diarrhœa in its relation to the bacterial content of food and dealt with cow's milk and food other than natural cow's milk, especially Nestlé's milk. He quotes Hope, Eustace Smith and Cautley to the effect that living bacteria are found in condensed milk, that such milk rapidly breeds bacteria even when still apparently fresh, and becomes unfit for the child's consumption, and that tins of condensed milk once opened are liable to decompose rapidly, especially in hot weather. His general conclusions are as follows:—

1. In proportion to the number of consumers, Nestlé's milk containing comparatively few bacteria is more frequently associated with diarrhœa than cow's milk in which the number of bacteria is phenomenally high.

2. In certain seasons cow's milk may be exposed to temperatures which favour a high bacterial count and yet not become a frequent source of diarrhœa.

3. The numbers of bacteria in preserved and natural cow's milk have no direct influence on the incidence of diarrhœa.

4. The great majority of cases of diarrhœa are due to the consumption of food which has been infected in the district in which the cases have occurred.

5. The infective matter thus conveyed to food is generally the excrement of some person suffering from diarrhœa.

6. The life history of house-flies and the facility with which they can convey the fæcal excrement of infected infants to food of the healthy suggest that the seasonal incidence of diarrhœa coincides with and results from the seasonal prevalence of flies.

Newsholme's views[5] coincide in large measure with those already detailed. He states that breast-fed infants have only one-tenth of the average proclivity of infants to fatal diarrhœa, and suggests that possibly toxic products of bacterial action may be operative both in fresh and condensed milk infection, and that the latter may be derived from the farm and not the domestic in all cases. Most of the evidence, however, is against this supposition. The whole question of the causation of infantile diarrhœa is yearly becoming of greater importance in Khartoum and other towns in the Northern Sudan, for while the native breast-fed child is not likely to be a sufferer, the infants of the lower class Europeans of various nationalities may and do fall victims to the disease. In the summer of 1907 there were a considerable number of cases of infantile diarrhœa in Khartoum. While these cases were doubtless due largely to contamination of milk, I have no doubt that improper feeding

[1] Nash, J. T. C. (May, 1906), *Practitioner*.

[2] Hewlett, R. T. (August, 1905), "The Etiology of Epidemic Diarrhœa." *Journal of Preventive Medicine*, p. 496, Vol. XIII.

[3] Griffith, J. P. C. (July 15th, 1906). *Therapeutic Gazette*.

[4] Sandilands, J. E. (January, 1906), "Epidemic Diarrhœa and the Bacterial Content of Food." *Journal of Hygiene*, p. 77, Vol. VI.

[5] Newsholme, A. (April, 1906), "Domestic Infection in Relation to Epidemic Diarrhœa." *Journal of Hygiene*, p. 139, Vol. VI.

* Article not consulted in the original.

Diarrhœa— *continued*

played a part, and in any case I question if flies were operative to any large extent as carriers of infection. The house-fly is at no time a great nuisance in Khartoum and is usually killed off in large numbers by the hot weather which begins in April as a rule. I am inclined to think that infected dust played a part, even though the conditions for sewage removal had been improved and there was little dysentery amongst the civil population. The chief cause, however, I believe to be the filthy conditions associated with the transport and distribution of the milk, which persist, despite efforts made to suppress them, and will persist until the measures which have been repeatedly recommended are put in force. This matter is dealt with under "Sanitary Notes," and so need not be discussed here at length :—

Turning now to symptoms and treatment :—

Batten[1]* classifies infantile diarrhœa as follows :—

1. Irritative, due to improper or undigested food. Stools bulky, green, sour and with curds.

2. Catarrhal, due to prolonged indigestion. Stools brownish-green with mucus and foul smell.

3. Ulcerative colitis. Rare. Blood and mucus stool.

4. Acute infective. Stools watery, often greenish, offensive. Choleraic symptoms.

The last is the "summer diarrhœa" type, and it is in this form that cerebral symptoms occur, due probably to toxic action.

As regards treatment in the very severe cases, liquor strychninæ hypodermically is said to be the best preventive of collapse, while ether and brandy hypodermically are contra-indicated. Transfusion with normal saline, followed by a hot bath and, when the rally has taken place, by stomach-washing are recommended as an effectual line of treatment. Sodium bicarbonate 2 grains to the ounce is used for the lavage. Rectal irrigation is useful, and Younge[2] speaks very highly of quassia infusion for this purpose, in doses of ½ to 1 dr. repeated every 3 or 4 hours as required. It is best given after a dose of castor oil to clear the bowel.

For feeding, albumen water, barley water, rice water, etc., all have their advocates, while in a case recorded by Myers[3]* nothing succeeded till a solution of gum arabic, 1 ounce to the pint, was given. This, at least, is a remedy easily obtainable in the Sudan.

As regards other forms of diarrhœa, Thresh[4] has recorded a widespread and serious epidemic due to a water supply having become polluted by washings from garden soil manured with road sweepings and the like. Such a condition is rare, but shows how carefully a public water supply should be guarded. In the Civil Prison at Khartoum cases of severe diarrhœa have occurred, due possibly to soakage of foul matters into a well. The area of cement round the mouth of the well had become cracked and broken, and it was the custom to wash vessels which had contained food on this spot. When the practice was discontinued the cases of diarrhœa no longer occurred. In some of them *B. pyocyaneus* may have been the exciting cause, as it was found post mortem in a case terminating fatally.

Castellani,[5] in Ceylon, found flagellates in the excreta of cases of diarrhœa. He describes three types and suggests that they were etiological factors in the production of the condition.

The rôle of *Balantidium coli* in diarrhœa is mentioned by Strong,[6] who thinks that man may sometimes derive this parasite from the hog. The encysted forms become dried and get blown about so that water or food may become contaminated. The diarrhœa is often associated with colic and persists until treatment is directed against the parasite.

The view that the hill diarrhœa of India is due to the presence of mica in water is criticised adversely by Maynard.[7] He regards it as due to liver congestion, the result of chill.

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[1] Batten, F. E. (January 3rd, 1906), *Clinical Journal*.

[2] Younge, S. H. (September 8th, 1906), "Treatment of Infantile Diarrhœa." *British Medical Journal*, p. 573, Vol. II.

[3] Myers, G. T. (June, 1906) *Medical Record*.

[4] Thresh, J. C. (November 28th, 1903), "Diarrhœa and Polluted Water." *Lancet*, p. 1519, Vol. II.

[5] Castellani, A. (November 11th, 1905), "Diarrhœa from Flagellates." *British Medical Journal*, p. 1285, Vol. II.

[6] Strong, R. P. (December, 1905), "The Pathological Significance of Balantidium coli." *Indian Medical Gazette*, p. 470, Vol. XL.

[7] Maynard, A. E. (January 20th, 1906), "Hill Diarrhœa." *British Medical Journal*, p. 141, Vol. I.

* Article not consulted in the original.

Diarrhœa—*continued*

Prout[1] has described an outbreak of dysenteric diarrhœa at Bathurst due to the fouling of drinking water by the excreta of locusts. These latter consisted of spindle-shaped bodies which were composed of the fibrous indigestible parts of the grass matted together, and also of the siliceous spicules found in many grasses. The result of their ingestion was a mechanical irritation like that induced by ground-glass poisoning.

Diphtheria. Most of the recent papers on this subject seem to deal with treatment, the preventive use of antitoxin, and the bacteriological aspect of the disease.

Sambon,[2] in an ingenious paper, seeks to prove a relationship between diphtheritic affections of man and those of the lower animals. He deals specially with avian diphtheria and states that if the diphtheria of fowls is transmissible to man, then the eggs of these birds must play an important part in its transmission, because diphtheritic patches have been found in the oviducts. The paper is interesting and suggestive, but is severely handled from the veterinarian standpoint by Mettam,[3] who states that the historical references and most of Dr. Sambon's quotations will not bear inspection for a moment. He agrees with the opinion of Friedberger and Fröhner relating to the transmission of animal diphtheria to man—it is a mere assumption due to ignorance of veterinary pathology.

In a discussion on "What is notifiable diphtheria?" Williams[4] divided the clinically mild and doubtful cases into three groups:—

1. Patients without ordinary clinical signs of diphtheria, not definitely ill and yet anæmic, with quickened pulses, nasal catarrh, and other local symptoms which bacteriologically prove to be diphtheria.

2. Cases with any of these lesions but with no general symptoms of ill-health.

3. Persons who are quite well and have no local lesions but by cultural tests are found to harbour diphtheria bacilli.

He is inclined to regard cases coming under groups 1 and 2 as requiring isolation and treatment, but as regards 3 he points out that there is no evidence that infected contacts can spread diphtheria until they have developed local symptoms.

Higley[5] describes a rapid method (fifteen minutes) of certain diagnosis by examination of stained smears from deposits or false membranes. The material for the smear is obtained by passing a looped needle flattened at the curve lightly over the false membrane. The stains used are: 1. Five drops Kuhne's carbolic methylene blue in 7 c.c. of tap-water. 2. Ten drops carbol fuchsine in 7 c.c. of tap-water. Method—Fix in usual way. Apply No. 1 for 5 seconds. Wash with tap-water and dry between filter paper. Apply No. 2 for one minute, wash, dry, and mount in balsam. Lœffler's bacilli then appear as dark red or violet rods, irregularly stained and often containing polar dots. The colour means nothing, the other points are characteristic.

Pennington[6] has a paper on the virulence of diphtheria organisms in the throats of healthy school children and diphtheria convalescents. He found that 10 per cent. of the former harbour in their throats bacilli morphologically indistinguishable from diphtheria bacilli. One half of these did not affect guinea pigs. About 30 per cent. of them were clearly attenuated, 14 per cent. moderately virulent. In the convalescent cases the great majority of the bacilli were highly virulent. His conclusions seem to be that, in healthy persons unexposed to infection, if diphtheria bacilli are present, they are usually non-virulent, that in healthy exposed people the organisms are markedly virulent and such persons are a fruitful source of infection, and that convalescents from diphtheria carry and disseminate virulent organisms as long as any remain in their throats, a period which may far exceed the duration of the clinical evidence of the disease. He submits that preventive measures should be based on these findings, but admits the practical difficulties of carrying such into effect. It is, therefore, evident that his views differ considerably from those of Williams.

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[1] Prout, W. T. (April 25th, 1908), "Unusual Cause of Dysenteric Diarrhœa in the Tropics." *Lancet*, Vol. I.

[2] Sambon, L. W. (April 18th, 1908), "The Epidemiology of Diphtheria, etc." *Lancet*, Vol. I.

[3] Mettam, A. E. (May 2nd, 1908). *Ibid.*

[4] Williams, P. W. (September 16th, 1905), "What is Notifiable Diphtheria?" *British Medical Journal*, p. 647, Vol. II.

[5] Higley, H. A. (May 20th, 1905), "Rapid Bacteriological Diagnosis of Diphtheria." *Epit. of British Medical Journal*, p. 80, Vol. I.

[6] Pennington, M. E. (Januuary 1st, 1907), "The Virulence of Diphtheria, etc." *Journal of Infectious Diseases*, p. 36, Vol. IV.

Diphtheria— *continued*

MacCombie[1] deals with the grave clinical significance of skin hæmorrhages in diphtheria. In pre-antitoxin days patients hardly ever survived more than two days after their appearance, and while they are now, thanks to antitoxin, rare, they almost invariably herald death within 4 or 5 days, though sometimes life is prolonged for a week or a fortnight. The fatal issue is due to toxæmia and cardiac failure, and persistent vomiting is often a marked symptom.

Ashby,[2] in a very well-illustrated paper, records an outbreak of milk-borne diphtheria associated with an ulcerated condition of the udders of cows. Like all such epidemics it was less severe and less fatal than the usual form.

Davies[3] has a very useful and practical paper with diagrams of highly magnified bacilli classified according to the types described by Westbrook, *i.e.* the granular, the barred and the solid types, each of which are sub-divided into varieties. He points out that school examination of contacts in infected classes is a much more rational procedure in urban communities than mere school exclusion without such examination, provided the possibility of home contacts is not forgotten. As regards "Carrier Cases," he quotes the conclusions of the Committee of Massachusetts Association of Boards of Health, which are as follows :—

1. It is impracticable to isolate well persons infected with diphtheria bacilli, if such persons have not, so far as known, been recently exposed to the disease.

2. It is not advisable, as a matter of routine, to isolate from the public all the well persons in infected families, schools and institutions.

The exceptions have to be made as a matter of expediency, in regard to wage-earners, business and professional men.

It is, however, advisable to keep the children in infected families away from day school, Sunday school and all public places.

Wage-earners may usually be allowed to continue their work, but teachers, nurses and others who are brought into close contact with children, and also milkmen, should not be allowed to do so.

In schools and institutions all infected persons, sick or well, should, if the infection is not too wide-spread, be separated from the others.

When diphtheria appears in a community which has for some time been free from it, it is advisable to isolate all persons who have been brought into contact with the patient until it shall have been shown that they are free from diphtheria bacilli.

Davies also suggests that the modified phenomena of the late stages of epidemic invasion may be due to an acquired immunity resulting from the prevalence of atypical forms of the diphtheria organism, especially Hoffmann's bacillus.

Rothe[4] describes a cultural method of distinguishing between true and pseudo-diphtheria bacilli. He uses a medium composed of a mixture of one part of neutral broth free from sugar and four parts beef serum. To this he adds ten parts of litmus, and dextrose or lævulose in a proportion of 10 per cent. of the whole. He finds that true diphtheria bacilli always attack the dextrose or lævulose and colour the litmus red, while, so far as is known, no pseudo-diphtheria bacillus has this combined action.

Graham-Smith[5] has a paper somewhat on the same lines. He found that most diphtheria-like organisms tested produce less acid than the diphtheria bacillus. Hoffmann's bacillus and diphtheria-like bacilli from the normal ear can be easily differentiated, since they form no acid. Any bacillus which acts on mannite or saccharose could also be easily differentiated.

Lewis[6] has a very useful paper on the bacteriological diagnosis of diphtheria. He notes that fallacies may arise, owing to fault on the part of (1) the clinician, or (2) the bacteriologist. As regards (1), the throat may have been treated with antiseptics prior to the application of the swab; the swab used may have been of wool impregnated with an antiseptic; the wrong locality of the throat may have been swabbed, this being a frequent

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[1] MacCombie, J. (December 22nd, 1906), "Exanthem of Scarlet Fever and some of its Counterfeits, and the Chemical Significance of Skin Hæmorrhages in Diphtheria." *British Medical Journal*, p. 1757, Vol. II.

[2] Ashby, A. (December, 1906), "A Milk Epidemic of Diphtheria associated with an Udder Disease of Cows." *Public Health*, p. 145, Vol. XIX.

[3] Davies, D. S. (March, 1907), "Diphtheria and Small-Pox: An Epidemiological Contrast." *Public Health.*

[4] Rothe (August 31st, 1907), "Beitrag für Differenzierung der Diphtheriebacillen." *Cent. f. Bakt.* I., *Orig.*, t. XLIV.

[5] Graham-Smith, G. S. (July, 1906), "The Action of Diphtheria and Diphtheria-like Bacilli on various Sugars and Carbohydrates." *Journal of Hygiene*, p. 286, Vol. VI.

[6] Lewis, C. J. (August, 1907), "The Bacteriological Diagnosis of Diphtheria." *Birmingham Medical Review.*

source of error. As regards (2), there may have been a perfunctory application of the swab to the serum; the temperature of incubation may have been wrong and not between 33° C. and 37° C., as is essential if the bacillus is to grow more rapidly than the accompanying cocci; the number of colonies examined may be too small, the slide may be greasy: the stain may be old or unfiltered; the staining may be careless; the examination may be too limited, *i.e.* sufficient fields may not be examined. **Diphtheria—** *continued*

After dealing with the characters of diphtheria bacilli and their classification, he concludes by stating that:—

The greater his knowledge of the circumstances of each individual case, the more valuable is the report of the bacteriologist. From a bacteriological standpoint alone a diagnosis of diphtheria, though generally reliable, is beset with difficulties. The bacteriological report must be a factor, and an important factor, in the decision, but the final judgment can only be made by the practitioner in conjunction with the medical officer of health.

Slater[1] reports a most interesting case of skin diphtheria of 3 years' standing. The original seat of the disease was the eyes, then the vulva became affected, the bacilli entered the superficial lymphatic circulation and produced a condition like herpes, possibly as the result of a toxic peripheral neuritis. Typical Klebs-Lœffler bacilli were isolated and no treatment had any effect until antitoxin was given, when the result was remarkable. The author does not say if this curious carrier case infected other people. Four other cases of skin diphtheria in the form of ulcers of the toe are narrated by Heelis and Jacob.[2] The condition at first resembled chilblains, but later a contact developed faucial diphtheria. Skin diphtheria, then, may in some measure explain the origin of certain obscure cases or even epidemics.

The question of treatment hardly falls to be considered here, but as it is sometimes difficult to obtain or store antitoxin in the Sudan, Leonard Williams'[3] strong advocacy of biniodide of mercury, given as a mixture containing the perchloride of mercury and iodide of potassium, may be cited. So may the use of 4 per cent. solution of formalin as a throat swab or gargle (Brunton[4])* and of formolyptol both as a spray and as an internal remedy in 2 minim doses (Rendle[5]).

Crookshank[6] advocates the hypodermic administration of adrenalin chloride and strychnine in severe cases of diphtheria marked by vomiting and cardiac depression. He employs tabloids, each containing $\frac{1}{200}$th of a grain of adrenalin chlorine and $\frac{1}{100}$th of a grain of sulphate of strychnine. One or two of these may be given every two, three or four hours. Even in desperate cases it may be of service, all food, other medicine and throat treatment being stopped when vomiting occurs.

The preventive use of antitoxin, however, calls for some brief notice, as, in such a country as this, provided the serum was available, it would constitute an important method of checking and controlling an epidemic.

Shackleton[7] records a school outbreak where antitoxin proved efficient as a prophylactic. The dose given was 2000 units of Burroughs Wellcome & Co.'s serum, or 1000 units of the Lister Institute serum. Norton[8] describes a somewhat similar experience, in which the results were most gratifying and there were practically no ill-effects.

Sittler[9]* has come to certain conclusions as to the length of immunity after injection of diphtheria antitoxin.

1. The immunity given by the prophylactic injections lasts from three to five weeks, if the children are not too often exposed to diphtheria in the interval.

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[1] Slater, A. B. (January 4th, 1908), "A Case of Diphtheria of the Skin of three years' duration treated by Antitoxin." *Lancet*, p. 15, Vol. I.

[2] Heelis, R., and Jacob, F. H. (March 10th, 1906), "A Series of Four Cases of Cutaneous Diphtheria." *British Medical Journal*, p. 556, Vol. I.

[3] Williams, Leonard (1907), "Minor Maladies." London.

[4] Brunton, T. L. (February 15th, 1906), *Clinical Journal*.

[5] Rendle, C. E. R. (February 18th, 1905), "Formolyptol in Diphtheria." *Lancet*, p. 460, Vol. I.

[6] Crookshank, F. G. (April 25th, 1908), "A Note on the Treatment of Diphtheria." *Lancet*, Vol. I.

[7] Shackleton, W. W. (Sept. 15th, 1906), "The Prophylactic use of Anti-Diphtheritic Serum." *Lancet*, p. 722, Vol. II.

[8] Norton, E. E. (July 13th, 1907), "The Prophylactic use of Antitoxin in Epidemic Diphtheria." *Lancet*, p. 85, Vol. II.

[9] Sittler, P. (September, 1906). *Jahrbuch f. Kinderheilk.*

* Article not consulted in the original.

Diphtheria—*continued*

2. Unimmunised children are much more susceptible to diphtheria than the children who have been immunised.

3. Catarrhal affections of all kinds, and wounds of the mucous membranes, predispose to diphtheria and tend to shorten the period of immunity.

4. The length of the period of immunisation is not increased by using doses larger than 500 units.

5. Certain children show a greater predisposition to diphtheria than others. It is advisable to isolate these children as thoroughly as may be, so as to avoid the necessity for too frequent injection of antitoxin.

In my experience cases of diphtheria, so far as Khartoum is concerned, are apt to crop up in October, when the summer has been dry. I have never seen an extensive epidemic nor have I ever been able to trace the disease to its source. Apparently it has been introduced from outside, and it is usually of a severe form associated with streptococcal infection. The type of diphtheria bacillus present has, as a rule, been what Westbrook would term Granular C. The disease is undoubtedly rare in the Sudan.

Disinfection. This is such a wide subject that it is difficult to pick out the papers most likely to be useful. Those selected will be found practical and to possess a bearing on sanitary work in the Sudan.

As regards the disinfection of ships, the Clayton process may be briefly described as one in which sulphur dioxide gas, produced by combustion of sulphur in a special apparatus, is driven into the lower parts of the holds which have been previously rendered air-tight. The air is extracted from the upper parts of the hold until all the air space is permeated with the gas to the extent of 10 per cent., the extracted air being passed over the heated sulphur in the furnace. One pound of sulphur is required for every 400 cubic feet of space. Three per cent. gas in the air is fatal to rats. There is no risk of fire, and the cost is £1 for every 100 tons gross register.

A Local Government Board report on the value of sulphur dioxide as a disinfectant and destroyer of rats is quoted in the *Lancet* for December 17th, 1904. It points out that the results achieved depend on whether the cargo is left in the hold or not. While, in the latter case, these are eminently satisfactory, rats, cockroaches and fleas being killed by a uniform diffusion of as little as ·5 per cent. of the gas, matters are quite different with the cargo *in situ*, owing to the slow penetrating power of sulphur dioxide. A suggestion is made that a small proportion of carbon monoxide (say 10 per cent. of "producer gas") should be added to the gas in the holds for the purpose of killing the rats. A later and similar report states that while carbon monoxide kills rats it fails to destroy mosquitoes and bacteria. Formaldehyde vapour, while destroying bacteria, spares rats and mosquitoes, has practically no penetrative power, and for its subsequent complete removal the disinfected material must be chemically treated.

A short account of the "producer gas" employed by Nocht and Giemsa in Hamburg is given in *Public Health* for September, 1905. The gas is generated by a current of air blown into a producer where coke is burned. The plant both introduces and removes the gas from the holds. It gives excellent results in the case of rats, while an apparatus enabling the generator gas to be mixed with formaldehyde vapour, so as to obtain simultaneously a disinfecting action, has likewise been provided. The cost is moderate. The prime cost of a large floating plant is about £2500, and if 100 vessels be treated a year the cost per vessel works out at about £3 or £4.

Sandwith[1] recently saw the apparatus at work. The gas took 12 hours to disinfect a large passenger steamer, but it is believed that all rats on a ship are killed after about ten minutes' exposure. The gas itself consists of about 8 per cent. carbon monoxide with a little carbonic acid and some 70 per cent. of nitrogen. The cost for disinfecting a moderate-sized steamer was £7. 10*s*. He does not mention the accompanying use of formaldehyde, but states that no harm results to the cargo.

Chloride of lime, if properly used, is so useful a disinfectant that a few papers on it may be quoted.

Hankin[2] worked on the subject in India and found that "specimens having the form of a coarsely granular powder keep longer than other specimens in which the material takes the form of adherent masses." His other conclusions are tabulated :—

1. Chloride of lime, when fit for use, has a strong smell of chlorine. If it has been kept in a hot climate for three months, the amount of available chlorine present will usually be about one-third of what it was originally.

[1] Sandwith, F. M. (November 30th, 1907). *Lancet*, p. 1535, Vol. II.

[2] Hankin, E. H. (September, 1904), "Chloride of Lime as a Disinfectant." *Indian Medical Gazette*, p. 351, Vol. XXXIX.

Disinfection —*continued*

After the lapse of the above period, the amount of available chlorine may be less and the substance will then be unfit for use.

2. Chloride of lime is readily attacked by various kinds of organic matter. Therefore, it is unsuitable for dealing with sewage or other large masses of putrefying material. On the other hand, it may be used with advantage in places where the infective material can only be embedded in small amounts of organic matter.

3. Owing to its deodorant properties and penetrative power, chloride of lime may be used in the interior of infected houses, both on the walls, on furniture, or on cement or stone floors.

4. Whitewash made in the usual way with quicklime is rendered far more active if half a pound of chloride of lime is added to every 7 gallons of the liquid.

5. Owing to the bactericidal power of chloride of lime under circumstances in which its action is not masked by the presence of an excess of organic matter, it is likely that it could be useful in the cleaning and disinfection of wells, either in place of, or mixed with, permanganate.

Owing to its unpalatable taste, it would, however, be necessary to pump out the well, preferably on the following day, before bringing the water of the well into use.

I have found that chloride of lime even when stored in a comparatively cool place in closed metal drums, rapidly deteriorates in the Sudan. A six months' old sample analysed by Dr. Beam was found to have only about 1 per cent. of available chlorine instead of the 30 per cent. which should have been present.

Klein[1] has a paper on the bactericidal efficiency of hypochlorites in the presence of organic matter. He experimented by adding chloros to urine, letting the mixture stand for an hour and then adding the typhoid bacillus. Owing to the previous action of the organic fluid on the disinfectant the co-efficient of the latter fell to 0·8. On the other hand, if chloros be added direct to typhoid infected urine, the co-efficient for chloros in a *watery* distribution of *B. typhosus* works out at 21·0.

It is worth noting that chloros, a valuable disinfecting agent at home, is not suitable for export.

A somewhat similar, but more elaborate, paper is that by Harris and Prausnitz[2] on fæces-urine emulsions used for testing disinfectants.

The disinfection of books is a practical question which often crops up. Formaldehyde is usually recommended, but Badia and Greco[3]* conclude that for a complete and proper disinfection of books the use of the autoclave is essential despite its drawbacks. On the other hand, Kister and Trautman[4]* find that if books of any kind are placed on a suitable stand with their leaves opened out so as to prevent more than six or eight pages sticking together and are then subjected to their process of formalin disinfection at an increased temperature in a vacuum, satisfactory results are obtained. No damage results, the only bad effect being a tendency for the leaves to curl. This is overcome by pressure.

In a country like the Sudan, where white residents are largely at the mercy of native servants, it is worth while knowing how readily and efficiently to disinfect ordinary table utensils. When one considers that such servants not infrequently suffer from venereal disease and other communicable disorders, the importance of such knowledge is apparent, though in actual life the necessity for its application would appear rarely to arise.

Beck[5] has studied the question, hitherto rather neglected, and finds that in most instances immersion in a 20 per cent. solution of carbonate of soda at a temperature of 50° C. suffices, but it will not serve in cases of infection by the *Tubercle bacillus*, and it is not easy to be sure of the temperature. Below 50° C. the action is ineffective, while a higher temperature damages table-knives, mounted forks, etc. Therefore immersion in alcohol at 60° C. for half an hour is recommended as an easy and reliable method. It is sometimes necessary to disinfect railway carriages. The formalin-permanganate method, in which formalin is poured upon crystals of permanganate of potash, is stated[6] to be the best. The proper proportions for use are one cubic centimetre of formalin to 0·5 gramme of the

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[1] Klein, E. (October, 1906), "The Bacteriological Efficiency of Hypochlorites in the presence of Organic Matter." *Public Health*, p. 27, Vol. XIX.

[2] Harris, C. E., and Prausnitz, C. (March, 1907), "The Determination of the Efficiency of Disinfectants." *Journal of the Royal Institute of Public Health*, p. 147, Vol. XV., No. 3.

[3] Badia and Greco, N. V. (August 7th, 1906). *Anal. del circ. Med. Argentino.*

[4] Kister and Trautmann, H. *Zeit. f. Tuberkulöse*, 1907, No. 6, p. 497.

[5] Beck, M. (August 7th, 1906), "Zur Frage der Disinfektion von Ess- und Trinkgeschirren." *Cent. f. Bakt. I. Orig.*, Bd. XLI., p. 853.

[6] *Lancet* (December 15th, 1906), p. 1675. "The Disinfection of Railway Carriages with Formaldehyde."

* Article not consulted in the original.

Disinfection —*continued*

permanganate, and from 200 c.c. to 500 c.c. of formaldehyde are required per 1000 cubic feet of air space. The presence of added water is unnecessary. The chief point about this method is the short exposure and the large quantity of gas evolved. It is also easily carried into execution and does not require elaborate apparatus.

Firth[1] draws attention to this method. The gas evolved consists of formaldehyde, water-vapour, carbon dioxide, and traces of formic acid, and the reaction is apparently expressed by the formula

$$4\,K\,Mn\,O_4 + 3\,H_2\,CO + H_2\,O = 4\,Mn\,O\,(OH)_2 + 2\,K_2\,CO_3 + CO_2.$$

The proportion of the two substances which gives the best results and the driest residue, is two parts of formalin to one part of permanganate. The method is effective, simple, rapid, and, by virtue of the inexpensive apparatus required, preferable to the older and more cumbersome methods. For a space of 2000 cubic feet, 285 grammes, or 10 oz., of the permanganate and 570 cubic centimetres, or one pint, of formalin are required, the reagents being mixed or added the one to the other in an ordinary galvanized-iron pail. The crystals, which are better crushed, are put in first, and then the formalin is poured on them. There is time for the operator to withdraw, and the period of disinfection should be six hours. Heat and moisture are essential for efficient disinfection. From 60° F. to 70° F. is a proper temperature, while it is well to render the air of the room moist in a dry country. One has employed this method on several occasions in Khartoum, and it appears to be efficient. The walls of the room to be disinfected are damped and the air sprayed with water before the gas is evolved. Firth's paper, which deals generally with disinfection by formaldehyde, contains much of interest, and he is inclined to urge the abandonment of so-called room disinfection altogether and confine attention to the infected person, his clothing and his bedding. The above method can be utilised for sterilising clothing in a very simple manner.

Recent work on plague has drawn attention to disinfectants capable of killing fleas.

Saigol[2] experimented with numerous chemicals. He found that petrol or benzine with cyllin or phenyle (in equal quantities) made up to 1 in 300, *i.e.* 1 in 1600 of both, were satisfactory. Actual contact with the fluid is necessary to kill the insects, though free use of the emulsion will drive out of a house those that escape actual contact. Female fleas are more resistant than the males. Both cyllin and phenyle emulsify petrol, but the former is preferable owing to its greater germicidal powers. The emulsion, for the making of which he gives directions, must be fresh.

Somerville[3] found that cyllin 1 in 400 and phenyle 1 in 250, and Jeyes' fluid 1 in 250, were efficient in five minutes, while a jelly of 80 per cent. petroleum with 20 per cent. whale oil soap used in a 3 per cent. solution is said[2] to be the best contact insecticide known. A 10 per cent. solution is absolutely certainly lethal for fleas. Hossack has done much work on this subject. He confirms Saigol's work with petrol and phenyle, but not as regards petrol and cyllin. This discrepancy was probably due to the difference in the samples used. He concludes that "the ideal for plague purposes would be a cyllin with the pulicidal power of the most potent samples of phenyle or phenyle with the germicidal power of a cyllin."

One may add a brief note on the disinfection of stools, and also of drain and water pipes, as it is sometimes difficult to obtain reliable information on these latter points. A good way of disinfecting cholera stools is to add together equal parts of fresh quicklime and water. Then dilute the slake lime so formed with three times as much water as has been previously used. Equal quantities of this mixture and cholera dejecta are thoroughly stirred together and allowed to stand for an hour, when all the vibrios are killed.

Fresh and good chlorinated lime in powder form, and in the proportion of two tablespoonfuls to a pint of cholera dejecta, is effective in twenty minutes. Strong izal, 5 per cent., or carbolic acid 1 in 10, with contact for two or three hours and thorough mixing, and a sufficiency of the disinfectant (rough guide = complete covering of the stool), are measures useful for enteric and dysenteric excreta. In typhoid bacilluria the urine may be diluted with half its volume of 1 per cent. formalin.

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[1] Firth, R. H. (April, 1908), "Disinfection by Formaldehyde." *Journal of the Royal Army Medical Corps*, Vol. X., No. 4.

[2] Saigol, R. D. (July, 1907), "The Flea-killing Power of various Chemicals." *Indian Medical Gazette*, p. 256, Vol. XLII.

[3] Somerville, D. (August, 1907), "Disinfectants against Fleas." *Indian Medical Gazette*, p. 316, Vol. XLII., No. 8.

For drain pipes a solution of ferrous sulphate 1 lb. to the gallon is recommended, while water pipes are disinfected by filling them with a 2 per cent. solution of carbolic acid for 24 hours and then flushing them out with pure water. **Disinfection** *—continued*

Dropsy *(Epidemic).* Our knowledge of this disease does not seem to have increased. Rogers[1] describes it as met with in India. The rash has to be remembered, while it would seem that the presence of the jerks and the absence of anæsthesia distinguish it from beri-beri. I have never heard of its being found in the Sudan.

Since the above was written, an important paper by Delany[2] has appeared. He was appointed to investigate the causation of beri-beri in Indian jails, but found that there was no true beri-beri in Eastern Bengal and Assam—the disease present being epidemic dropsy. He believes this latter to be a specific infectious or bacterial disease, which is conveyed from person to person by bed-bugs. His reasons for looking upon it as a bacterial disease are: (1) Its epidemic character; (2) the initial fever; (3) the rash or rashes; (4) the local or house infectiousness; (5) the sudden disappearance of the disease when infected houses are vacated. The bed-bug theory is supported by (1) the well-known manner in which the disease affects households; (2) its close association with the sleeping places of affected persons; (3) the presence of bug-infested bamboo stools (morahs) in an infected district; (4) the benefits resulting from evacuation of infected jails and houses. He recommends the latter method combined with bug prevention and bug destruction as likely to prevent and eradicate the disease.

Pearse,[3] the Health Officer of Calcutta, is inclined to believe that beri-beri and epidemic dropsy are one and the same disease due to a specific organism, but Delany, in the paper just quoted, enters very fully into this question and tabulates the particulars in which the diseases resemble and differ from each other.

The diseases resemble one another in the following manner:—

(*a*) Both occur mostly in epidemics.

(*b*) The knee jerks are altered in each.

(*c*) Dropsy of various degrees occur in both.

(*d*) There is considerable cardiac disturbance in each, dilatation and heart murmurs being present, or palpitation and dyspnœa only.

(*e*) In each disease the pericardium, pleura and peritoneum may contain fluid.

(*f*) In each disease there is frequently œdema of the lungs.

(*g*) Cutaneous sensation is disturbed in both diseases.

(*h*) Hyperæsthesia occurs in both.

(*i*) In each disease motion is frequently disturbed or interfered with.

(*j*) And in each disease death occurs with distressing dyspnœa and orthopnœa.

But the diseases differ as follows:—

(*a*) Knee jerks in beri-beri are at first and for a brief period (rarely over 48 hours) increased and painful, and then lost in probably more than 95 per cent. of cases.

In epidemic dropsy knee jerks are diminished or lost in no more than 3 per cent. of cases.

(*b*) Anæsthesia is a marked feature of beri-beri and will be found in practically every case either in small patches or over extensive areas. In epidemic dropsy cutaneous sensation is lessened over the dropsical areas and not in patches otherwise than over dropsical areas; but in this disease, though cutaneous sensation is diminished, it is not lost, and probably is only so diminished from mechanical interferences with nerve termini by the effused fluid.

(*c*) In beri-beri true paralyses occur, with toe drop, wrist drop, paraplegia or paralysis of all four limbs. In epidemic dropsy various forms of paresis are simulated by mechanical obstruction around, joined by the effused fluids; the very weight of a swollen limb may cause a difficulty in using it. An ataxic gait is simulated owing to the swollen legs, and this may be more apparent when the external genital organs are swollen.

But in beri-beri a characteristic symptom is the presence of varying degrees of paralysis in cases that have no dropsy whatever (dry beri-beri), and this occurs, according to Hunter and Koch of Hong Kong, in quite 50 per cent. of the cases, these cases having besides the characteristic patchy anæsthesia.

(*d*) The hyperæsthesia differs in the two diseases, being present in the dropsical skin and subcutaneous tissue when gently pinched in epidemic dropsy; but in beri-beri, the muscles are painful on moderate deep pressure in œdematous and non-œdematous parts alike.

(*e*) Some few cases of epidemic dropsy are found to undergo a general emaciation and so simulate the atrophic stage of beri-beri in which the muscles atrophy to such a degree that the patients look like living

[1] Rogers, Leonard, "Fever in the Tropics." London, 1908.

[2] Delany, T. H. (May, 1908), "Epidemic Dropsy or Beri-beri in Eastern Bengal." *Indian Medical Gazette.*

[3] Pearse, F. (March 2nd, 1908), "On the Identity of Beri-beri and Epidemic Dropsy." *Journal of Tropical Medicine and Hygiene.*

Dropsy— *continued*

skeletons. But these cases of emaciation are able to move their limbs about in bed, though they are feeble. In any large outbreak of beri-beri these cases of atrophy with extensive and severe paralysis are present in quite large numbers and are often bedridden for many months.

(*f*) A marked feature of beri-beri is the sudden deaths that occur in addition to the distressing deaths with dyspnœa and orthopnœa, such as also occur in epidemic dropsy. These sudden deaths occur not alone in cases with paralysis and dropsy, but in persons apparently well, or who have but the mildest symptoms.

(*g*) There is some leucocytosis and anæmia (diminution of hæmaglobin) in epidemic dropsy, but in beri-beri anæmia is not present.

(*h*) Of minor importance are the presence of rashes (subcuticular mottling and staining along the course of superficial veins), with dry skin and slight desquamation and initial fever in epidemic dropsy.

(*i*) Lastly, the symptoms of beri-beri are essentially those of peripheral neuritis, and the central nervous system is unaffected in every case. (Hunter and Koch, Manson, Braddon, Wright).

Reaney[1] advances arguments against Delany's view that bed-bugs may be the carriers.

Dust. Few subjects are of more importance than this in the Northern Sudan, and hence a paper on Dust Problems[2] merits attention. "A road to be dust proof," it says, "should have a smooth, impermeable, enduring surface, and a hard foundation which will not work out through the top; such a road can be built by using slag taken hot from the furnace and dipped in tar until soaked from surface to centre, after a method invented by Mr. Hooley, of Nottingham, under the name of 'tarmac.'" While this is good for new roads it would not pay to lift and relay those already existing, and in Khartoum no slag is available. Palliatives, known as "Westrumite" and "Akonia" are said to be inexpensive, and their effect on dust much more enduring than mere watering. They were considered for the Khartoum streets, but the cost was found to be prohibitive.

Dysentery. A vast deal of literature has accumulated on this important subject and it is no easy matter to pick out the papers most profitable for review. No doubt many have been missed, but it is hoped those selected will prove useful.

Waters[3] brings out very strongly the influence of soil contamination as a factor in the spread of dysentery, and especially in camps, jails and institutions. He cites South African experience, the rôle played by flies and the filthy habits of native prisoners. He also points out that men who have suffered much from malaria are very prone to dysentery, and that previous dysenteric attacks also predispose to the disease. He found that hard, out-door labour, necessitating exposure, favoured dysentery, and he mentions the substances used by malingerers to produce a condition like the disease. As regards the blood state, a general increase in the small lymphocytes was noted.

Hewlett[4] reviews the findings of Schaudinn as regards the differences between *Entamœba histolytica* and *Entamœba coli* (*vide infra*), and alludes to Musgrave's and Clegg's work on Amœbiasis and the cultivation of amœbæ. The most important point to which he refers is the apparent necessity for symbiosis with bacteria for the growth of the amœbæ. In this connection he cites Lesage, who found, along with *Entamœba histolytica*, a bacillus which he termed the *Paracolon bacillus*.

McWeeney[5] mentions briefly the vegetative and sexual cycles of both forms of amœbæ and refers to Schaudinn's classical and careful experiment of feeding a young and healthy cat with meat and milk infected solely with the small, brown spores of *Entamœba histolytica*. The cat died of dysentery and showed characteristic ulceration of the large intestine, while crowds of amœbæ were found in the ulcers and penetrating the wall of the gut.

The amœboid stage of the parasite was found incapable of transmitting the disease. It would seem that the disease is not propagated by amœbæ introduced per os. The older experimenters had often succeeded in producing infection by the introduction of amœbæ per rectum, but, as this can hardly be realised under natural conditions, it is to the dried-up spore-containing fæces present in dust and water that we must look for the propagation of dysentery. These spores can be conveyed by flies and can be blown about by the wind.

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[1] Reaney, M. F. (July, 1908), "Epidemic Dropsy." *Indian Medical Gazette.*

[2] "Dust Problems." *British Medical Journal*, p. 1763, December 31st, 1904.

[3] Waters, E. E. (December 1st, 1903), "Dysentery." *Journal of Tropical Medicine*, p. 363, Vol. V.

[4] Hewlett, R. T. (April, 1905), "Pathogenic Amœbæ and their Cultivation." *Journal of Preventive Medicine*, p. 237, Vol. XIII.

[5] McWeeney, E. J. (March 25th, 1905), "On the Relation of Parasitic Protozoa to each other and to Human Disease." *Lancet*, p. 783, Vol. I.

Dysentery—*continued*

Fearnside[1] has a paper on jail dysentery, and one may note two of his conclusions. (1) Mud banks (sleeping places) should be abolished and plank beds substituted, as the former are insanitary and apt to become septic. (2) Association wards should be done away with and the cellular system introduced, as the segregation thereby obtained tends to check the spread of infectious disease.

O'Kinealy[2] points out the frequent association of oral sepsis, evidenced by bleeding and unhealthy gums and jail dysentery and diarrhœa. He believes that careful attention to prisoners' teeth and gums is very necessary.

Matthews[3] mentions an outbreak at Aden due to the inhalation of contaminated dust.

Newell[4] classifies dysenteries as: (1) Catarrhal. (2) Acute, specific or bacillary. (3) Amœbic. (4) Spirillary. (5) Mixed. (6) Chronic. This is useful, but takes no account of fluxes due to the malarial parasites, *Balantidium coli* and *Trichomonas*. We know also that there is a dysentery associated with kala-azar, and verminous dysentery forms a class by itself. Newell also lays stress on the influence of wind-blown infected dust.

Spirillary dysentery has been mentioned. Dantec[5]* made a clinical study of this form, which is easily distinguished from the bacillary type by the absence of any temperature rise. The liver is not affected, and the proper treatment, rapidly effectual, is by antiseptic enemata.

I have seen one case of animal spirillary dysentery in the Sudan. It was found by Captain Olver in a native dog belonging to myself. There was no rise of temperature, but the stools were full of blood and mucus and the animal rapidly emaciated.

Dopter[6] insists on the unity of bacillary dysentery. Under this term he would include the so-called pseudo-dysenteries and dysenteries of infants and aliens.

Gauducheau[7] reports that when trying to reproduce abscess of the liver in a dog by a portal injection of pus from a human hepatic abscess he brought about a fatal amœboid dysentery. This is a matter of considerable interest. Indeed, from the post mortem appearances in a case of multiple liver abscess which came under my notice, I suggested[8] that, in some instances, hepatic abscess may precede a dysenteric affection of the large bowel.

Vedder[9]* classifies the characteristics of the dysenteric and normal amœbæ as follows:—

Entamœba histolytica (Dysenteriæ)	*Entamœba coli*
Size	
25-30 microns (not a distinguishing feature)	10-20 microns
Shape	
Usually some other shape.	Spherical when resting.
Colour	
Greenish.	Opaque greyish.
Protoplasm	
Ectoplasm and entoplasm easily distinguished	Ectoplasm and entoplasm distinguished with difficulty.
Ectoplasm very refractive.	Ectoplasm not refractive.
Ectoplasm finely granular.	Ectoplasm homogeneous.
Entoplasm coarsely granular.	Entoplasm finely granular.

[1] Fearnside, C. F. (July, 1905), "Dysentery in the Prisons of Madras Presidency." *Indian Medical Gazette*, p. 241, Vol. XL.

[2] O'Kinealy, F. (July, 1905), "The Relation of Oral Sepsis to Dysentery." *Indian Medical Gazette*, p. 250, Vol. XL.

[3] Matthews, E. A. (July, 1905), "The Etiology of Dysentery, with Notes on Treatment." *Indian Medical Gazette*, p. 253, Vol. XL.

[4] Newell, A. G. (July, 1905), "Dysentery: Its Varieties and Causes, Summarised and Criticised, with a Note on Treatment and Prevention." *Indian Medical Gazette*, p. 257.

[5] Dantec, "*La Caducée*." December 17th, 1904.

[6] Dopter, C. (January 15th, 1906), "La Dysenteric Bacillaire, Discussion sur l'Unité Specific." *Bulletin de l'Institut Pasteur*, p. 49.

[7] Gauducheau, A. (January 15th, 1906), "On Experimental Reproduction of Amœbic Dysentery by Intravenous Inoculation of Pus from a Hepatic Abscess." *Journal of Tropical Medicine*, p. 52, Vol. IX.

[8] Balfour, A. (November 21st, 1903), "A Case of Multiple Liver Abscess." *Lancet*, p. 1425, Vol. II.

[9] Vedder, E. B. (March 24th, 1906). *Journal American Medical Association.*

* Article not consulted in the original.

Dysentery— *continued*

Pseudopodia	
Large and easily distinguished. Certain ectoplasm and entoplasm.	Hard to distinguish. Entirely ectoplasm.
Vacuoles	
Many.	Often absent. Never more than one.
Nucleus	
Often absent. When present its structure hidden except in stained specimens. Nuclear membrane not well defined. Changes position markedly.	Almost invariable, with well-defined nuclear membrane and other structure. In moving, organism retains relative position.
Red Corpuscles ingested	
Many.	None observed.
Motility	
Great progressive motility.	Often absent, or, when present, of limited extent and short duration.

Lösch, quoted by Manson, gives very similar characteristics, and in addition mentions—

Multiplication	
In the intestine by fission and budding. On hard fæces or outside the body resistant spores formed without encystment.	In the intestine by binary fission and also by multiple fission into 8 amœbulæ. On hard fæces and outside the body encystment and formation of 8 amœbulæ.
(These develop when swallowed).	(These are set free when swallowed).

Musgrave and Clegg,[1] in a long paper on the cultivation and pathogenesis of amœbæ, oppose Schaudinn's views, and believe that the name *Amœba coli* (Lösch) should still be retained to represent those amœbæ which are found in human intestines. They do not believe in differentiating between *E. coli* and *E. histolytica* for the following reasons:—

Amœbæ found in the stools of so-called healthy people do not always conform to the requirements for *E. histolytica*. Cultures of amœbæ answering more nearly the description given for *E. coli*, can by methods described in this paper (M. & C.) be made to produce ulcerative colitis in man and monkeys and abscesses of the liver, omentum, spleen and lungs in monkeys. In cultures, single species of amœbæ are often found which are characterised by possessing a combination of some of the features which have been described as distinctive for different species. Amœbæ from many extraneous sources, and presumably saprophytic, may be cultivated on artificial media, and with such cultures ulcerative amœbic colitis may be produced in man and animals, and abscesses brought about in the liver, lung, omentum, spleen and muscular tissues of animals.

These conclusions are, however, challenged by Vedder,[2] who points out that many of them are based on fallacies and faulty working methods, and concludes that the criticisms are not well founded.

Ashburn and Craig[3]* have worked at the presence of amœbæ in healthy persons, American soldiers in the Philippines. They examined 100 cases. In 72, *E. coli* was present; in 2, *E. dysenteriæ*. None of the 72 had dysentery or diarrhœa at the time of examination, nor had they ever been on the sick list owing to these diseases. The two men with *E. dysenteriæ* appeared well, but were found to have dysenteric symptoms and were eventually invalided for chronic amœbic dysentery. Their interesting conclusions are as follows:—

In the Philippine Islands a very large proportion of white men are infected with *E. coli*, and such infection does not result in symptoms of diarrhœa or dysentery; in many of the cases the amœbæ disappear but in the large proportion *E. coli* may be found even after the lapse of nine months, during which time the infected individuals have remained in perfect health as regards dysentery or diarrhœa.

We also conclude that *E. coli* differs very markedly from *E. dysenteriæ* as regards morphology, and that it is possible to distinguish these two species of amœbæ by their morphological characteristics as observed in fresh specimens of fæces. We do not believe that the very large proportions of infections with *E. coli* which we have

[1] Musgrave, W. E., and Clegg, M. T. (November, 1906), "The Cultivation and Pathogenesis of Amœbæ." *Philippine Journal of Science*, p. 909, Vol. I.

[2] Vedder, E. B. (June 1st, 1907), "Is the Distinction between Entamœba Coli and Entamœba Dysenteriæ Valid?" *Journal of Tropical Medicine and Hygiene*, p. 190.

[3] Ashburn, P. M., and Craig, C. F. (September, 1907). *The Military Surgeon*, p. 222, quoted in *Indian Medical Gazette*, December, 1907.

* Article not consulted in the original.

demonstrated can be explained logically by the theory of "latent infections," but only, as we have stated in a previous report, "by the fact that the non-pathogenic *E. coli* is the organism present in these cases instead of the pathogenic *E. dysenteriæ*." **Dysentery—** *continued*

Dr. Wenyon, working in Khartoum at cases of all kinds in the Military Hospital, found that in the great majority *E. coli* or its cysts were present. These cysts were found in recently passed fæces, and he has shown[1] that in mice and monkeys the formation of similar cysts commences in the cæcum. In cases of dysentery these cysts of *E. coli* were also present, while the small cysts of *E. histolytica* were not seen. Further reference to this work will be found in Dr. Wenyon's report.

Birt[2] has worked at South African dysentery. He isolated Shiga's bacillus in 26 out of 55 cases examined. Amœbæ were only found once. The method of examination adopted may be detailed. Wash a fragment of blood-stained mucus in sterile salt solution. Transfer to a second lot and shake vigorously. Place on Drigalski-Conradi medium, taurocholate neutral red, crystal violet, lactose agar, lactose litmus agar or ordinary agar (+ 25 Eyre's scale). Next day transfer more delicate colonies to Doer's modifications of Barsikow's medium consisting of nutrose, mannite, litmus and water. Shiga's bacillus leaves this unchanged. The *colon bacillus* curdles it and turns it pink. Sub-cultivate on agar and, if Gram-negative rods obtained, put up in sedimentation tubes with the patient's serum, normal human serum and the serum of an animal immunised with Shiga's serum.

Highest dilution noted for clumping was 1 in 600, the usual 1 in 20–50. The agglutinating power was not of long duration.

Blackham[3] has written a very excellent paper on tropical dysentery, chiefly from the bacteriological standpoint, at the close of which he states that the various strains of *B. dysenteriæ* isolated by Shiga, Flexner, Vaillard, Harris, Firth, etc., are simply varieties of the same organism. There are also non-pathogenic pseudo-dysentery bacilli which act on carbohydrates unaffected by Shiga's and the other pathogenic bacilli, and fail to produce enteritis in animals. The dysentery organisms will live on clothing for at least three weeks and are said to maintain their virulence in *damp* soil for months. Spread on bread crumbs or similar articles of food, they survive for about a week. They are not very readily destroyed by heat or by weak solutions of perchloride of mercury or the higher phenols. The Widal reaction is often poorly marked, but is of some value, and can usually be obtained within two weeks of the onset of symptoms.

The character of the stools alone is not to be considered as a test of the presence or absence of dysentery. A group of maladies of varying severity come under the term dysentery, ranging from the acute dysentery of armies to the simple infective diarrhœa of infants and adults.

Blackham believes that "in the tropics all cases of diarrhœa should be treated with the same precautions as if they were manifest cases of dysentery, and in hospital should invariably be isolated and their stools sterilised in some simple form of steriliser or by means of disinfectants."

Duncan,[4] in a useful paper, describes the different types of stool met with in cases of dysentery, and concludes by considering the indications obtainable from the different appearances of the stools in dysentery as regard *prognosis*.

1. A good result can be foreshadowed in those cases in which are passed mucus with minute fæcal lumps, stained or not with blood, and in which the blood and mucus disappear; the ordinary fæcal characters will soon manifest themselves.

2. The prognosis is of evil omen: (*a*) according to Sir Joseph Fayrer, in the cases in which pulpy stools without blood or mucus are passed; (*b*) where fluid fæcal matter is from time to time passed throughout the illness, the prognosis is unfavourable, inasmuch as these characters of the stools show the disease to be extensive, and affecting chiefly the upper part of the large, as well as in some cases part of the small, intestine; (*c*) where the stools, in conjunction with the symptoms that are laid down as characterising the true amœbic dysentery, are present, the prognosis is again unfavourable on account of the high mortality that is said to attend this form of the disease; (*d*) the prognosis is of the worst possible

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[1] *Archiv. für Protistenkunde*, Suppl. I., 1907.

[2] Birt, C. (March 31st, 1906), "Dysentery in South Africa." *Lancet*, p. 904, Vol. I.

[3] Blackham, R. J. (December 1st, 1906), "Tropical Dysentery." *Lancet*, p. 1493, Vol. II.

[4] Duncan, A. (May 2nd, 1904), "The Stools of Dysentery and the Prognostic Indications derivable from them." *Journal of Tropical Medicine*, Vol. VII.

Dysentery— *continued*

character where the stools consist of blackish-red or blackish fluid, with a horribly putrescent odour, and of bits of gangrenous tissue.

Dopter[1] records three cases of amœbic dysentery, in which all ordinary methods of treatment failed, but which were speedily cured by lavage with a 1 in 100 creosote wash. He suggests that the creosote does not merely act locally, but, being absorbed, reaches and acts upon the *Amœba dysenterica* in the tissues and in localities where it cannot be affected by other medicaments.

Vincent[2]* believes that in water-borne epidemics the amœba is more frequently the cause than the bacillus. He investigated the length of life of *B. dysenteriæ* in various waters and the action of antagonistic saprophytes, and concludes that water is not a suitable medium for the bacillus. At the same time, it lives a long time in frozen water with light excluded, which perhaps explains the outbreaks and frequency of epidemics in cold countries.

Billet[3]* has described a special form of Trichomonas which he terms *T. dysenteriæ* as distinct from *T. intestinalis*, and which he believes plays a part in the production of tropical dysentery.

So far as Khartoum is concerned there can be little doubt that polluted surface soil played an important part in the only dysentery epidemic—a slight one—which has visited the town. Reference to this and other points with relation to dysentery in the Sudan will be found under "Sanitary Notes" (Third Report).

As regards the treatment of dysentery, one must distinguish between measures suitable for bacterial dysenteries and those useful in amœbic cases. The treatment for the former class has been revolutionised by the introduction of appropriate sera, and considerable literature has accumulated on this most important subject.

Blackham[4] in the first place gives a table for the differential diagnosis of Amœbic and Bacillary Dysentery :—

Amœbic	*Bacillary*
1. Always chronic in its course.	1. Acute in onset and running a rapid course in nearly all cases.
2. Pyrexia rare.	2. Pyrexia common.
3. Toxic symptoms not present except where there is liver abscess.	3. Toxic symptoms usually present.
4. Liver abscess occurs in about 16 per cent. of cases (Curry).	4. Liver abscess never occurs.
5. Small intestine frequently attacked.	5. Disease confined to large intestine.
6. According to Krause and Kartulis, undermined ulcers present.	6. Ulcers usually found on surface folds of intestine.

Personally I very much doubt if bacillary dysentery is always confined to the large intestine. In the Second Report of these Laboratories I recorded a rapidly fatal case of a disease exactly like dysentery where, post mortem, all that was found was a comparatively small area of the small intestine acutely inflamed and presenting an appearance like a measles rash. I had no opportunity of examining this case bacteriologically, but it was either bacillary dysentery or some hitherto unrecognised, infective, inflammatory process.

The main points brought out by Blackham as regards treatment are: (1) Value of opium in doses of gr. $\frac{1}{4}$ to gr. $\frac{1}{3}$ of morphine hypodermically. (2) Clear soups are better than milk; and weak chicken broth, whey and egg albumen may be given till the tongue cleans. (3) Stimulants rarely necessary; when required try a teaspoonful of brandy in a tablespoonful of coffee. (4) Value of preliminary dose of castor oil with or without 15 or 20 minims of Liquor Opii Sedativus. (5) Medicinal treatment lies between use of sodium sulphate or of calomel The latter is given in gr. $\frac{1}{2}$ doses every hour for twelve hours during the day, stopped at night, and repeated in the same way during the second and third days. Bismuth should be given after the calomel for 3 or 4 weeks. (6) The specific serum treatment is valuable and should be tried. (7) In sub-acute and chronic cases in the tropics, where good

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[1] Dopter, C. (February 12th, 1908), "Traitement de la Dysenterie Amibienne par la Créosote." *Bull. de la Soc. Path. Exot.*

[2] Vincent, H. (June, 1906). *Revue d'Hygiène*, t. XXVIII, No. 7.

[3] Billet, A. *La Caducée*, August 17th, 1907.

[4] Blackham, R. J. (February, 1908), "The Treatment of Dysentery." *Journal of the Royal Institute of Public Health*, p. 77, Vol. XXIV.

* Article not consulted in the original.

nursing is available, lavage is valuable. (8) Any morbid condition of the blood must be attacked, *i.e.* malarial infection by quinine and diminished alkalinity by lactate of sodium. **Dysentery—** *continued*

For amœbic dysentery, ipecacuanha is stated to be the sovereign remedy. Thirty to forty grains, presumably with the usual precautions, are administered at first and the dosage diminished every night. Then castor oil with or without opium is exhibited, very small doses being given. Finally, simaruba with aromatics and an intestinal antiseptic, such as salol or salicylate of bismuth, conclude the cure.

Vaillard and Dopter[1] report most excellent results with the anti-dysenteric serum prepared in the Pasteur Institute, Paris. It was found to greatly lessen mortality, to diminish the severity of the symptoms, and to hasten recovery. They insist on early administration, the giving of sufficient dosage, regulated by the gravity of the case, judged by the numbers of stools in the 24 hours and general symptoms of intoxication. In cases of moderate severity, 20 c.c. suffice. In very severe cases, up to 100 c.c. may be given repeatedly each day till improvement results.

Sandwith,[2] in a review of the whole subject, mentions chronic "dysentery carriers," and the rare occurrence of mixed bacillary and amœbic cases. He details the serum rules (Shiga) in Japan. These are (1) in mild cases the serum is injected in one dose of 10 c.c.; (2) in cases of average severity, a second time after an interval of from six to ten hours; and (3) in severe cases repeated twice daily for two or three days. The serum is derived from horses repeatedly inoculated subcutaneously with an emulsion of Shiga's bacillus in a normal saline solution which has been heated to 60° C.

By medical treatment alone, patients recover in 40 days or die on the eleventh day; by the serum treatment, they recover in 25 days or death is postponed till the sixteenth day.

A polyvalent serum is likely to prove the best, there being so many different strains of dysentery bacilli. Ipecacuanha for amœbic dysentery was found disappointing in Egypt, and calomel is not recommended, but the fractional method of dosage is not considered. The sulphate of magnesium or sodium treatment gave good results in Egypt. Rules for lavage are mentioned, one pint of fluid increased rapidly to two pints being the quantities usually employed, and the value of this treatment in certain instances before cases have become chronic is emphasised.

Castellani[3]* has tried the opsonic treatment in a case of chronic dysentery with marked success. The Kruse-Shiga bacillus isolated from the stools was used in the preparation of the vaccine.

Drake,[4] writing from Assam, reports very favourable results from the administration of gr. 5 yellow santonin with dr. 2 of olive oil. Unfortunately he does not say what type of dysentery was present. It is quite possible that it may have been the verminous variety, which would explain the beneficial action of an anthelmintic drug.

Forster[5] has a paper on the vaccine-therapy of dysentery. The vaccine employed consists of a dead emulsion of *B. Shiga* in normal salt solution to which 0·5 per cent. of carbolic acid has been added. The emulsion is prepared from 24-hour agar slope cultures and is killed by heating to 60°-63° C. in a water bath for twenty minutes.

Stephen[6] records a case of old-standing dysentery in a British Officer treated with this vaccine. Perfect cure apparently resulted after three inoculations, although the patient had previously been practically incapacitated for work during a period of five years.

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[1] Vaillard and Dopter, C. (April 26th, 1907), "La Sérotherapie dans le Traitement de la Dysenterie Bacillaire." *Ann. de l'Inst. Pasteur*, t. XXI.

[2] Sandwith, F. M. (December 7th, 1907), "Hunterian Lecture on the Treatment of Dysentery." *Lancet*, p. 1589, Vol. II.

[3] Castellani, A. (1907). *Archiv. für Schiffs und Trop. Hyg.*, Bd. XI., Heft. 3.

[4] Drake, D. J. (November 1st, 1907), "The Treatment of Dysentery by Yellow Santonin." *Journal of Tropical Medicine*, p. 351, Vol. II.

[5] Forster, W. H. C. (June, 1907), "A Preliminary Note on the Application of Vaccine-Therapy to Dysentery." *Indian Medical Gazette*, p. 201, Vol. XLII.

[6] Stephen, L. P. (October, 1907), "Case of Old-Standing Dysentery treated by Vaccine-Therapy." *Indian Medical Gazette*, p. 375, Vol. XLII.

* Article not consulted in the original.

Gillit[1] also describes cases successfully treated at Midnapore Central Jail. The mortality before this line of treatment was adopted was 5·9 per cent., since its introduction only 0·9 per cent. The number of cases recorded is not very large—140 all told—but there seems no doubt as to the efficacy of the treatment.

Elephantiasis. Castellani[2] has used thiosinamin in the form of Merk Fibrolysin (a water soluble combination of thiosinamin with sodium salicylate) in the palliative treatment of elephantiasis. After bandaging and massaging of the infected parts the drug is injected in doses of 2 c.c. every day or every other day for about a month. Then the fibrolysin is stopped and rubber bandaging or ordinary bandaging again resumed for a week or ten days. Thereafter another course of thirty or more injections is given, and so on as required. In suitable cases he believes this treatment may prove useful.

Christophers,[3] in a paper entitled "What is really known of the cause of elephantiasis?" points out the grounds on which the assumption that it is due to the presence of *Filaria nocturna* in the lymphatics is based. He shows that deductions drawn from geographical relationship and race incidence may be faulty. He also states that we can only say that *presumably* elephantiasis is due to blockage of the lymph channels. One perhaps is on firmer ground when noting the association of elephantiasis with other diseased conditions, some of which are undoubtedly due to filaria, such as varicose lymph glands, lymph scrotum, etc. Doubtless the active inflammation, and even hæmorrhage produced by the worms, have more to do with the pathological conditions than the mere presence of the worms themselves. He lays stress on the difficulty of "explaining how with so complex a collateral circulation the blocking can ever be so complete as to lead to the terrible conditions one so frequently sees, and the need for actual and accurate observation on the disease, especially as regards the blocking of glands by undeveloped embryos and a consideration of the localisation of the blocking."

Prout[4] has a long paper on the rôle of filariæ in disease production, dealing, however, solely with *F. loa* and *F. nocturna*. As regards the latter and its relations to elephantiasis, Prout announces himself a sceptic with reference to Manson's theory, and, especially as regards localised elephantiasis, is on the look-out for a specific micro-organism, gradually spreading by the lymphatics from the periphery.

In the discussion[5] on this paper its author's views were rather severely criticised. Low contended that filaria was at least one of the causes of elephantiasis. Carnegie Brown held that though elephantoid disease was certainly due to filaria, the relation of the latter to elephantiasis had not been proved. Basset Smith, however, mentioned a case of apparently recent elephantoid disease in which no filaria were found. Manson discriminated between tropical and non-tropical forms of elephantiasis and elephantoid disease, and stated that the filarial doctrine of elephantiasis, which was too readily accepted, was now threatened with too hasty a rejection. The journal must be consulted for full details.

As regards the Sudan the question of elephantiasis is briefly considered under the heading "Filariasis" (*page* 70).

Enteric Fever. Probably the most valuable recent contribution to our knowledge of Enteric Fever, from the tropical standpoint, is the work by Roberts.[6] One cannot refer to it here at any great length, but of special importance to those working in the Sudan are the conclusions regarding the liability of the native Indian to the disease. Roberts believes that the Indian possesses a natural immunity of a two-fold nature. It is in part racial, due to anatomical differences in the intestine, for, as he points out, both the large and small intestines in natives are in many instances considerably longer than in Europeans. He cites a case of a Mohammedan in whom both guts combined totalled 50 feet. Further, the intestinal walls in natives are thicker and more muscular, and Peyer's patches are not so

[1] Gillit, W. (January, 1908), "Notes on Forster's Vaccine Treatment of Dysentery." *Indian Medical Gazette*, p. 12, Vol. XLIII.

[2] Castellani, A. (August 1st, 1907), "Note on a Palliative Treatment of Elephantiasis." *Journal of Tropical Medicine and Hygiene*, p. 250, Vol. X.

[3] Christophers, S. R. (November, 1907), "What is really known of the Cause of Elephantiasis?" *Indian Medical Gazette*, p. 404.

[4] Prout, W. T. (April 1st, 1908), "On the Rôle of Filaria in the Production of Disease." *Journal of Tropical Medicine and Hygiene*, p. 109.

[5] Discussion on above paper in *Journal of Tropical Medicine and Hygiene* of June 1st, 1908.

[6] Roberts, E., "Enteric Fever in India and in other Tropical and Sub-Tropical Regions." Thacker Spink & Co., Calcutta, 1906.

Enteric Fever— *continued*

much in evidence. The other factors operative are diet, habits, general surroundings and adaptation of the human organism to the disease causes which are most prevalent.

As regards habit and dietary, he gives an interesting comparative table which in large measure applies as much to the Sudan as to India, though in towns like Khartoum there is no doubt that the habits and dietary of certain classes of the natives, and especially the servant classes, has altered considerably within the past few years, and will continue to do so as a direct result of increased prosperity, a higher standard of comfort and association with, and imitation of, Europeans:—

The Native	*The European*
Cold and dry.	Hot and fluid.
Bulky and coarse. Much waste.	Concentrated and soluble.
Vegetable grains. Cereals and pulses; large cellulose content. Low proteid and fat content.	Animal food with high proteid and fat.
Very partially cooked, plain and monotonous from day to day.	Thoroughly cooked and sophisticated. Very mixed and varied.
Meals infrequent, twice daily with long fasts.	Meals frequent, 4 or 5 times a day.
Mastication generally good.	Faulty in extreme.
The majority eat to live.	More often live to eat.
Life and work in open air.	Sedentary, indoor.
Fæcal evacuations twice daily, large 10-12 oz.; completer by squatting.	Small 5-6 oz.; constipation rife. Purgatives.
Strain on stomach and large bowel.	Strain on stomach and small intestine.

The influence of these dietaries and habits on toxic putrefactive processes in the intestine is discussed in a very interesting manner.

As regards the diseases prevalent, the author lays great stress on the liability of the Indian to dysentery and other bowel complaints apart from enteric fever, and thinks that the reaction of the tissues against *B. dysenteriæ* in its various forms may confer local immunity against the closely allied *B. typhosus*. Indeed, when he considers the question from the bacteriological point of view, he is inclined to favour the theory that *B. coli* under favourable conditions may develop in the intestinal canal into the true *B. typhosus*. It must be admitted that this is a very engaging theory and that one sees cases of what are probably *B. coli* infections which very closely resemble early enterics. Indeed, one has felt that if such cases had not been promptly treated with calomel and appropriate dieting, they would, in all probability have passed into a condition almost indistinguishable from typhoid fever. On the other hand, there is no definite proof that this ever occurs, while Roberts' views on the rarity of enteric fever in the native Indian are opposed by Rogers,[1] who finds that the disease is widely prevalent save in Eastern Bengal and Assam, where there is a heavy and continued rainfall. Further, he states that the clinical picture in natives is precisely the same as that in Europeans. Thus, while Roberts believes that European troops chiefly obtain infection in the cantonments themselves, owing mainly to faulty conservancy methods, Rogers maintains that the native bazaars also present foci of infection. His conclusions are chiefly based on the evidence obtained by the application of the Widal test, but Roberts' book is so carefully compiled, his reasoning seems so accurate, and his conclusions are so well supported by statistics from other tropical countries, that there is much to be said for his attitude on the subject. It is, of course, possible that the native suffers from a mild and unrecognised form, but, so far as Khartoum goes, I do not think this is the case, for, if the disease were at all common, the conditions governing our water supply would assuredly have led to epidemic prevalence amongst the susceptible European population. This matter, however, will be further discussed under "Sanitary Notes" (Third Report). Rogers[2] explains the difficulty by pointing out the low incidence of typhoid amongst persons over the age of 25 years in India. He believes this explains the comparative rarity of the disease in the native army and in jails, which, he says, led Roberts to conclude that natives of India were relatively immune because the majority of those in the native army and in jails are over this age. He finds the disease not uncommon in native children and in the poor Europeans of Calcutta reared under the same conditions as the native.

However, there seems little doubt that the disease, in *epidemic form*, is rare amongst natives in India, while in the Sudan I believe it is, so far, rare in any form.

Stock[3*] believes that enteric fever is a common disease of tropical regions and mentions

[1] Rogers, L., "Fevers in the Tropics," 1908.

[2] Rogers, L. (August, 1907), "The Incidence of Typhoid Fever on Civilian Europeans and on Natives in Calcutta." *Indian Medical Gazette*, p. 291, Vol. XLII.

[3] Stock, P. G. (January, 1908), "The Etiology of Enteric Fever." *Transvaal Medical Journal*.

* Article not consulted in the original.

that in South Africa the Kaffirs suffer from it much more frequently than is supposed and tend to scatter infection broadcast.

Considerable importance now attaches to the question of typhoid carriers, *i.e.* persons who have recovered from the disease but harbour the specific germ in their bodies and are in a condition to infect those with whom they or their excreta come in contact.

Levy and Kayser[1]* record the results of the bacteriological examination of the body of a person who was known to have been a typhoid fever "carrier" during life. The patient, who was in an asylum, must have harboured bacilli for several years and had re-infected herself from the gall bladder or bile ducts. She died of typhoid sepsis, but during life the bacilli were present in her stools and she had undoubtedly been the cause of several small epidemics.

Kayser had previously recorded two cases in 1906, one in the person of a female baker who infected every new employee at the bakery which she owned, and the other in a female engaged in the milk trade, who was apparently responsible for the outbreak of an epidemic due to infected milk in which 17 cases were involved with two fatalities.

These and other instances are referred to by A. Ledingham and T. C. S. Ledingham[2] in a paper dealing with cases of enteric fever which kept cropping up in a Scottish lunatic asylum and which were traced to the presence of three typical typhoid carriers. They state that the bacilli probably vegetate in the gall bladder, from which they are intermittently ejected into the intestine, and make it clear that anyone found to be a typhoid carrier should be kept constantly under bacteriological supervision. They also suggest that possibly many typhoid epidemics would be avoided if the excreta of recovered typhoid cases (especially female cases) were examined systematically (say once a month) up to six months after recovery. A point they mention which is worth noting is that typhoid stools submitted for bacteriological examination should on no account be mixed with urine, as the latter markedly inhibits the growth of intestinal organisms on the plate. They also give a useful bibliographical table.

In the *Lancet* for January 23rd, 1908, allusion is made to an outbreak in a Home for Inebriates, which was also traced to a typhoid carrier, and mention is made of Dudgeon and Gray's work, which resulted in the finding of typhoid bacilli in bone lesions 3½ years after an outbreak of enteric fever.

Dean[3] has drawn attention to the case of a typhoid carrier of twenty-nine years' standing, and details the bacteriolgical method he employed in recovering *B. typhosus* from the stools. A general review of the subject will be found in the copy of the journal in which Dean's paper occurs.

Forster,[4]* impressed by these discoveries, has put forward a new theory as regards the pathogenesis of typhoid. Because the bacillus is regularly found in the gall bladder during, and often for a long time after, the disease, because it is usually not found in the fæces in the early stages, while Conradi has found it in the blood during the incubation period, and because if one injects typhoid bacilli into the circulation of animals they are excreted into the bile, Forster concludes that the bacilli taken into the stomach and intestines with food and drink do not multiply there but pass into the circulation from which they are excreted into the liver and bile. He believes the bacilli which appear in the stools after the end of the first week of the fever are derived from this source and from the intestinal ulcers.

The same is more or less true of paratyphoid infections. The occurrence of "carriers" is explained by the fact that bile plus proteid matter (say inflammatory products) constitutes a good medium for the *B. typhosus*. Most enteric patients cease to be carriers after two to six weeks, but about 2 per cent. go on excreting bacilli for several or even many (20 or more) years. The majority of these are women, females being more liable to diseases of the gall bladder than men.

As showing the great hygienic importance of these carriers, Forster presents some very interesting statistics. Of 386 cases investigated, 77 (20 per cent.) were due to infection

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[1] Levy, E., and Kayser, H. (December 11th, 1906). *Münch Med. Woch.*

[2] Ledingham, A., and Ledingham, T. C. S. (January 4th, 1908), "Typhoid Carriers." *British Medical Journal*, p. 15.

[3] Dean, G. (March 7th, 1908), "A Typhoid Carrier of Twenty-nine Years' Standing." *Lancet*, Vol. I.

[4] Forster, J. *Münch. Med. Woch.*, 1908, No. 1, p. 1.

* Article not consulted in the original.

from "carriers," 117 (30 per cent.) to contact infection, 45 (12 per cent.) were indefinite, and the remainder due to food or water infection. Gall bladder symptoms should be looked for in persons who have recovered from enteric fever, and if the stools are to be examined it is well to previously administer a laxative or cholagogue. **Enteric Fever—** *continued*

Preventive measures are very difficult. Strict cleanliness and the regular employment of disinfectants are indicated. Cholagogues, intestinal antiseptics, the introduction of lactic acid bacilli and anti-typhoid immunisation have all been used in order to try and dislodge the bacilli, but in vain. Some obstinate cases have apparently been cured by cholecystotomy or cholecystectomy. (Dehler).

For much interesting information regarding carrier cases and the etiology of the disease, the reader may be referred to the papers[1] read at a recent discussion on typhoid fever. One of these, by Hamer, puts forward his bold and heterodox views regarding the etiological rôle of *B. typhosus*. He does not think this organism necessary for the production of typhoid fever, but his ingenious arguments do not appear to have convinced many of his hearers.

One may next pass in review the several recently devised methods for facilitating the diagnosis of enteric fever.

These may be classed as: *(a)* clinical. *(b)* bacteriological. The former may be divided into the ophthalmo-reaction test and the observance of certain special symptoms, the latter into the agglutination test, the blood culture test and the recovery by new, special methods of the specific organism from the stools or urine.

After the introduction of the ophthalmo-diagnostic method for the diagnosis of tuberculosis, it occurred to Chantemesse[2]* to try a like reaction in the case of enteric fever. He killed cultures of *B. typhosus* by heating them; dried, powdered, and emulsified them in water. This emulsion was sedimented and centrifugalised till it was only slightly opalescent, and an active principle (a soluble toxin) was then obtained by precipitation with absolute alcohol. This precipitate is dried and keeps well. 1/50th of a milligramme of this powder is the dose, and it is used in solution, being instilled into the conjunctival sac of the patient. If the latter has enteric fever and gives the Widal test, an inflammatory reaction occurs which lasts for several days. Positive results were obtained in 63 cases of enteric fever and negative results in 50 patients who were not suffering from typhoid and did not give the Widal test. If rabbits be inoculated subcutaneously with typhoid bacilli and then after 48 hours tested in this way, they are found to give the reaction while healthy rabbits yield negative results.

Philipowicz's sign is regarded by Regis[3]* as pathognomonic. It consists of a more or less definite yellow coloration of the palms and soles. It is said to be most common in children, less so in women and least of all in men. It commonly appears during the first week, vanishes when convalescence is established, but reappears if a relapse occurs. The same author mentions Bernard's sign, which consists in the presence of two or three small swellings, varying in size from a filbert to an almond, and to be made out by careful palpation in the right iliac fossa. They are believed to be due to swollen Peyer's patches in the lowest part of the ileum, lie parallel to the long axis of the colon, and are from a half to one inch distant from each other.

Rolleston[4]* has drawn attention to the value of the condition of the abdominal reflex as a diagnostic and also as a prognostic sign. He says:—

1. The abdominal reflex is affected in a very large number of cases of enteric fever, the percentage of cases in which it is entirely lost exceeding those in which its normal activity is only diminished.

2. From its absence under the age of fifty being confined to certain nervous disease and acute abdominal conditions, notably appendicitis and enteric fever, the absence of the abdominal reflex in a given case of continued pyrexia in any patient below fifty is of considerable value.

3. The comparatively transient nature of the affection of the abdominal reflex in enteric fever is a striking contrast to the more chronic affection of the knee and ankle-joints in diseases associated with peripheral neuritis, *e.g.* diphtheria.

4. Return of a lost reflex, and, *a fortiori*, resumption of its normal activity, are a valuable indication of commencing convalescence, and often correspond with lysis and characteristic changes in the fæces and urine.

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[1] *Proceedings of Royal Society of Medicine* (April, 1908), Epid. Section, Vol. I., No. 6, p. 169.

[2] Chantemesse. *Deutsche Med. Woch.*, No. 39, and *Bull Acad. Médecine*, July 23rd, 1907.

[3] Regis, L. (July 4th, 1906). *Medical Press.*

[4] Rolleston, J. D. *Brain*, 1906, p. 99.

* Article not consulted in the original.

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ever—
continued

5. The objective sign of return of the reflex is often associated with the return of the subjective feeling of ticklishness normal to the individual.

6. In reappearance of pyrexia in convalescence, the condition of the abdominal reflex is a valuable index of the nature of the pyrexia. (That is to say, its disappearance or its becoming sluggish would point to a relapse.)

7. No constant relation exists between the condition of the abdominal reflex and that of the tendon reflexes.

8. The frequency, degree and duration of impairment of the abdominal reflex are, as a rule, in direct proportion to the age of the patient.

Another clinical aid is the recognition of leucopænia. Horder[1] records a case in which the bacilli were demonstrated in the blood in the absence of the Widal test and where there was a marked leucopænia, the white cells numbering 1400 only. Here the leucopænia suggested enteric fever and the further examination which led to the diagnosis being established. Gennari[2]* has specially investigated this subject. He concludes that in the early stage of typhoid, leucopænia, if present, is a valuable aid to diagnosis, and all the more so because at the beginning the Widal reaction is often negative. Leucopænia may be met with in miliary tuberculosis, but it is rarer in these cases. If any complications occur in the course of the typhoid a comparative leucocytosis may ensue, but apart from this, leucopænia or a normal leucocyte count is the rule. Leucopænia seems more often present in bad cases. In 55 of Gennari's 106 cases the differential leucocyte count showed no departure from the normal in 19, while in 36 there was diminution of the polynucleated cells, increase of the lymphocytes, absence of eosinophile cells and increase in the mononuclears.

Passing now to bacteriological methods, we find the value of Ficker's modification of Widal's test mentioned[3] in an article taken from a Berlin medical paper. While it is quite reliable and very convenient, it has the disadvantage that the reaction is useless in the early stages of the fever and is not obtained much before the end of the second week of the disease.

Numerous papers testify to the value and usefulness of the Widal test, although the discovery of the paratyphoid fevers has, to some extent, interfered with it. Rogers[4] devotes a page to this subject. He regards complete reactions up to 1 in 100 by the microscopical method, with a time limit of one hour as almost absolute evidence of an actual attack of typhoid or immediate convalescence from one. Such reactions are exceptional in the first week of the disease, and only found by a single examination at later dates in three-fifths to two-thirds of the cases. Reactions in lower dilutions are highly suggestive and should lead to further testing after a few days have elapsed, while any case at all resembling typhoid in which a negative reaction is got should also be re-tested in five to seven days' time. It must also be borne in mind that *repeated negative reactions throughout may be obtained in undoubted and often very severe typhoid*, so that when clinically there are any good reasons for looking on a case as one of typhoid, a negative Widal should be allowed little or no weight against the clinical diagnosis. Where it is most valuable is in enabling very mild and abortive cases to be recognised and properly treated, and preventive measures against the spread of the disease being taken, but it *still remains only an additional aid in forming a diagnosis*, which in the majority of cases can be usually made from a study of the temperature curve, pulse rate and other clinical characters before a reliable Widal reaction can be obtained. This is an excellent précis of the case, and in the Sudan one has met with the exceptional cases mentioned. Rogers' technique for the Widal test can also be recommended, and one has adopted it in preference to Rostoski's method previously employed. The latter author[5] discusses the conditions other than enteric which are apt to give the Widal reaction. Chief amongst these is jaundice, and hence it may be found present in Weil's disease. A case of puerperal fever has also been known to give a positive Widal, while proteus and staphylococci can give rise to an agglutinin acting on typhoid bacilli. Direct culture from the blood has come to the fore of late.

Castellani[6] describes his so-called "dilution method," which consists in adding small quantities (a few c.c.) of blood to comparatively large quantities (300 c.c.) of faintly

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[1] Horder, T. J. (January 19th, 1907), "A Case of Typhoid Fever Diagnosed by Blood Culture in the Absence of the Agglutination Reaction." *Lancet*, p. 168, Vol. I.

[2] Gennari, C., *Ref. Med.* (March 16th, 1907). Quoted in Epit., *British Medical Journal* of June 15th, 1907.

[3] November, 1906, "The Diagnosis of Typhoid by Ficker's Test." *British Medical Journal*, p. 451, Vol. II.

[4] Rogers, L., "Fevers in the Tropics." London, 1908.

[5] Rostoski, "Manual of Serum Diagnosis," Translated by Bolduan, 1904.

[6] Castellani, A. (May 5th, 1906), "Early Diagnosis of Typhoid Fever." *British Medical Journal*, p. 1071, Vol. I.

* Article not consulted in the original.

alkaline beef broth or peptone water, incubating at 37° C. and sub-cultivating the organisms which develop. The novelty of the method lies in the great dilution of the blood. **Enteric Fever—** *continued*

Conradi[1]* introduced the bile method, now generally employed. He took the blood from the ear in a capillary pipette containing a little fluid. The liquid is then transferred into a small glass tube containing 2 c.c. to 3 c.c. of a peptone glycerin mixture (10 per cent. peptone and 10 per cent. glycerin with ox bile), and this process of transference is continued till the ear ceases to bleed. Proportion of blood to the bile mixture is 1 to 3. The tube containing the blood and bile mixture is then incubated at 37° C. for ten to sixteen hours, and cultures finally made on Drigalski-Conradi agar plates. The diagnosis is usually established in from 26 to 32 hours.

Meyerstein[2]* uses the active bile constituents, the biliary acids, as his medium for increasing the number of bacilli present in the blood. The substance can be prepared by a simple method, and the results are said to be more quickly obtained and to be more reliable than those given by the Widal test.

It has also been found that typhoid bacilli can be cultivated from the blood-clot of specimens sent for a Widal reaction (Müller and Graff[3]*), and this has been confirmed, good results being got by spreading the clot on an Endo or Drigalski plate. It would seem that the colonies cannot be obtained from the serum.

A better and simpler way is to put the clot into a tube containing 5 c.c. of fresh ox bile which has been sterilised in a steam oven or by boiling in water; the tube, after cooling, being plugged with wool as usual. Incubate clot and bile for a night at 37° C. and plate out as mentioned. The bile is said to dissolve the blood and set free the organisms. This method is likely to be useful in the Sudan where blood is often sent from a distance and sometimes arrives in such a condition that it is difficult to obtain sufficient serum for the ordinary Widal test.

Zeidler[4] records his results with the bile method, using 30 drops of blood to 5 c.c. of sterilised ox bile, incubating at 37° C. and plating out on agar or the malachite agar of Lœffler. He concludes that from the fourth day of the fever onwards, both in mild and severe cases, the presence of the specific bacillus in the blood can be demonstrated, and that this method is superior to all others, being the most certain, the most speedy and, when positive, absolutely reliable, while it is at least as simple as the Widal test.

Coleman and Bunton[5]* employ a mixture of ox bile 90 c.c., glycerin 10 c.c., and peptone 2 grammes. This is distributed into small flasks, 20 c.c. in each, and sterilised. Three flasks are used for each examination, about 3 c.c. of blood being run into each. The flasks are incubated, and next morning streaks from each are made over the surface of a litmus-lactose-agar plate. The authors believe that *B. typhosus* is present in the blood in every case of typhoid fever, and that failure to recover it is due to error in technique. The bacilli seem to disappear about the time the temperature falls. They are found equally in the blood, but not with the same persistence in the mild as in the severe cases. To produce typhoid fever it seems necessary that the bacillus must not only be present in the body and growing, but that it should be in a situation whence it has free access to the blood. The bacillæmia does not constitute a true septicæmia, but represents an overflow of bacilli from the lymphopoietic organs.

Although I have not seen it advocated, save by its author, and have had no personal experience of it, mention may be made of Poeppelmann's[6]* method. After cleaning the finger and pricking it with a sterilised needle, a large drop of blood is squeezed out on a clean slide. Portions of this are transferred to, and spread out on, other slides previously cleaned with alcohol and ether and sterilised. After the films have dried, the slides are placed in R. May-Grünwald solution.

This fixes and stains at the same time and within 2 to 6 minutes. Wash in distilled water for about one minute and dry rapidly before a fire or between blotting paper. The

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[1] Conradi, H. (January 11th, 1906). *Deut. Med. Woch.*

[2] Meyerstein, W. *Münch. Med. Woch.*, 1908.

[3] Müller and Graff, H. *Münch. Med. Woch.*, 1906, No. 2.

[4] Zeidler, G. (August 20th, 1907), "Zur Frage der Typhusanreicherung mittels der Gallenkultur." *Cent. f. Bakt. I. Orig.*, Bd. XLIV.

[5] Coleman, H., and Bunton (June, 1907). *American Journal of Medical Science.* Quoted in Epit. *British Medical Journal*, April 25th, 1908, p. 68.

[6] Poeppelmann, P. W. *Deut. Med. Woch.*, 14th June, 1906, quoted in Epit. *British Medical Journal*, December 1st, 1906.

* Article not consulted in the original.

Enteric Fever— *continued*

examination is made under high powers (1000 diams.). The bacilli are coloured blue, but in their case the stain varies in intensity. Failure to stain may be due to disintegration. Very large numbers may be found. The sources of error are discussed and are to be avoided by scrupulous cleanliness, filtering the stain, taking care not to decolorise for more than one minute in water and to dry rapidly.

Still dealing with the bacteriological diagnosis, we come to the methods in vogue for isolating *B. typhosus* from the urine and fæces. These are very numerous and in many cases complicated, so that brief reference to some of the more recent must suffice.

Lœffler[1]* now recommends a special medium containing nutrose 1 per cent., peptone 2 per cent., glucose 1 per cent., chemically pure malachite green 1 per cent. of a 2 per cent. solution, and normal salt 1·5 per cent. In this mixture *B. typhosus* produces a true coagulation, the whole mass becoming solid save for a clear green liquid comparable to the whey of clotted milk. The appearance with *B. coli* is quite different, owing to the fermentation of the sugar and production of gas. The presence of malachite green is not essential. It merely hastens the reaction.

It was Lœffler who discovered that malachite green added to nutrient gelatin or agar inhibited the growth of *B. coli* but not that of *B. typhosus*. On such plates, however, only colonies in proportion to the bacilli actually present in the material examined can develop. The number of such, as in shell-fish, water, milk, etc., may be very limited, hence Klein[2] has devised a true "enrichment" process. He used for this purpose fluid media to which bile salt was added, making indeed a malachite green bile salt broth. He records good results with this medium, and the method of preparation will be found detailed in his paper.

The method of Lentz and Tietz may be described. They crush up the stool in an equal quantity of 0·8 per cent. NaCl solution and plate out on the surface of a malachite green plate (malachite green No. 1, Höchst, 1 to 6000 of agar). Incubate for 24 hours at 37° C. If no colonies of *B. typhosus* be found, suspend the surface growth of the plate in about 8 c.c. to 10 c.c. of broth, and inoculate from the uppermost layer of this broth, which has been allowed to stand for some time in the plate which is sloped.

Rivas[3]* believes that the frequent failure in detecting *B. typhosus* in infected water supplies is largely due to faulty laboratory technique. He shows that litmus, Parietti's solution, Drigalski-Conradi medium, the Endo medium and others, have actually a germicidal effect upon the bacillus whatever their value may be as means of differentiation.

The viability of *B. typhosus* in sterilised and unsterilised soils has recently been investigated by Mair,[4] who finds that :—

1. The typhoid bacillus can survive in natural soil in large numbers for about 20 days and is still present in a living condition after 70 to 80 days.

2. There is no evidence that the typhoid bacillus is capable of multiplying and leading a saprophytic existence in ordinary soil.

3. In some samples of soil, but not in all, the typhoid bacillus dies out much more rapidly (in 11 days) if the soil has previously been subjected to sterilisation by steam under pressure. This is apparently due to the production of bactericidal substances during sterilisation.

Enteric fever has of late received special consideration from a military standpoint.

Harrison[5] has dealt with the preponderating importance of dust, flies, and personal infection in hot countries, the difficulties of enforcing suitable conservancy methods and, as a result, the necessity for the exclusion of typhoid carriers from a force when it takes the field and the necessity for general anti-typhoid inoculation. He also advocates special depots for typhoid convalescents.

Davies[6] has an important paper on direct contagion, that is to say, personal infection. He admits that it may play an important part in the spread of the disease, and tabulates a

[1] Lœffler, F., "Zum Nachweise und Zur Differenzial-diagnose der Typhusbacillen mittels der Malachitgrün nährboden." *Deut. Med. Woch.*, No. 39, 1907.

[2] Klein, E. (November 30th, 1907), "A Contribution to the Bacteriological Analysis of Materials Polluted with the Bacillus Typhosus." *Lancet*, p. 1519.

[3] Rivas, D. (1908). Quoted in *Lancet*, June 27th, 1908.

[4] Mair, W. (January, 1908), "Experiments on the Survival of *B. Typhosus* in Sterilised and Unsterilised Soil." *Journal of Hygiene*, p. 37, Vol. VIII.

[5] Harrison, W. S. (November 23rd, 1907), "Enteric Fever in War." *Lancet*, p. 1463, Vol. II.

[6] Davies, A. M. (August 31st, 1907), "Enteric Fever, its Spread by Personal Infection, and Preventive Measures on Active Service." *British Medical Journal*, p. 505.

* Article not consulted in the original.

Enteric Fever—*continued*

lengthy list of thorough preventive or protective measures. Most of these are the same as those laid down for any dangerous, infectious disease. We specially note that all remains of food supplied to patients, and not consumed, should be destroyed, that everything which enters an enteric ward, books, journals, even empty soda-water bottles, should be regarded as infective and treated accordingly, that all utensils and apparatus for use in enteric cases should be kept separate and apart and specially marked, and that nursing attendants should be specially detailed, fed and housed apart, and, while not absolutely isolated, should wash and change their clothing before associating with the rest of the staff.

Rules are given for the procedure to be followed on admission of a case to hospital (disinfection of kit, bedding, etc.), when convalescence is established (use of urotropine, examination of the stools, etc.), also in slight cases apt to be unrecognised (provision of quarantine or segregation camps, etc.). The paper then goes on to deal with enteric under active service conditions, and, while space forbids further quotations, it may safely be said that it is well worth the perusal of all in medical charge of troops and those responsible for the health of men in camps, as for example the camps of the Survey Department in the Sudan.

Another useful paper on the same lines is that of Caldwell.[1] He recommends shallow and narrow latrine trenches 1 foot in depth and 1 foot in breadth, and the direction that men should straddle across these to prevent fouling of their edges. Straton, whom he quotes, advocates the use of a 2 per cent. solution of crude carbolic acid in latrine buckets to keep away the flies.

Statham,[2*] dealing with etiology, mentions that there are a number of allied but distinct species of bacteria, which may produce disease indistinguishable from typhoid fever, and that most of the varieties of bacteria composing the typhoid colon group, are found in apparently healthy animals, while many diseases amongst domestic and other animals are caused by bacteria which may produce typhoid in man. He also refers to the fact that typhoid has been induced in human beings by eating the improperly cooked flesh of such diseased animals.

As regards anti-typhoid inoculation, Leishman[3] describes the preparation of the new modified vaccine. The bacilli are only subjected to 53° C., the minimum temperature which ensures their death within one hour. The results with this vaccine have been most encouraging. In one regiment out of a strength of 509, 147 were inoculated. Sixty-two cases of enteric with eleven deaths occurred, all amongst the uninoculated with the exception of two, both being men who had refused the second inoculation; both of these men recovered.

This author[4] also records the utterances of Chantemesse as regards the remarkable results obtained by the use of his curative serum. Chantemesse stated that he had never lost a patient in whom the treatment was commenced during the first seven days of the disease. Especially noticeable is the fact that the spleen appreciably enlarges after the serum injections—Chantemesse attributes to this an important part in the origination of the beneficial changes which are found in the blood itself (leucocytosis, increase of mononuclears, rapid reappearance of eosinophiles).

With reference to treatment, perhaps the most suggestive of recent papers is that by Young[5] on the dietetic management of cases. After prolonged trial he has entirely disregarded the "antiseptic" method of treatment in all its forms, being convinced that when diarrhœa (or tympanites) occurs the only true remedy is *a careful revision of the dietary*. In a properly dieted case these troublesome symptoms should not arise, for the diet should fulfil the following requirements:—

1. It must be such that no solid residue, and certainly none of the least irritating character, enters that part of the testinal tract where the local lesions are situated.
2. It must be such that fermentation of such a kind as to generate flatus does not take place.
3. Inasmuch as the whole of the digestive functions are below par, it must be one which is readily digested and assimilated.
4. It must be such that the various tissues are provided with proper material for the renewal of that waste of substance and vitality common to all prolonged fevers, and especially such pyrexial conditions as are accompanied by the circulation of toxins.

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[1] Caldwell, R. (August 31st, 1907), "On Enteric Fever during Active Service." *British Medical Journal*, p. 513.

[2] Statham, J. C. B. (January, 1908), "The Complex Nature of Typhoid Etiology, etc." *Transvaal Medical Journal.*

[3] Leishman, W .B. (March, 1908), "The International Congress of Hygiene, Berlin." *Journal of the Royal Army Medical Corps*, Vol. X., No. 3, p. 247.

[4] Leishman, W. B. (March 23rd, 1907), "Anti-typhoid Inoculation in the Army." *Lancet*, p. 806.

[5] Young, M. (September, 1906), "The Dietetic Treatment of Enteric Fever." *Public Health*, p. 686.

* Article not consulted in the original.

Enteric Fever— *continued*

In a properly dieted case constipation takes the place of diarrhœa, but this tendency can be corrected by the use of sanatogen. Young mentions a list of foods devised to satisfy the cravings of the enteric patients for an ampler dietary:—

1. Benger's and Mellin's foods, made with or without milk and fortified with cream, are of temporary value. So are jellies, bread crumbs, isinglass or rusks in beef-tea, and light puddings.

2. Bread jelly made by thoroughly soaking stale bread, pressing out the water and allowing the pulp to simmer gently for one or two hours. Strain through muslin and allow filtrate to set. Two tablespoonfuls of the jelly suffice for one feed.

3. Raw meat pulp carefully prepared and given in the form of little balls to be eaten with a rusk.

4. Junket made in the usual way and given if desired with cream or brandy.

5. Suet puddings, given after the temperature has been normal for a few days. The suet must be shredded in thin slices and all the fibre removed. Wheat flour with an equal quantity of maize should be used. The latter contains little gluten. Cook well and serve with sweet sauce or gravy.

6. Fish, best in the form of whiting.

7. Modified milk diets. Swithinbank and Newman's rules for boiling milk to prevent alteration in flavour and formation of scum are given.

(i.) Use an ordinary double milk pan, or a smaller covered saucepan containing the milk placed inside a larger one containing the water.

(ii.) Let the water in the outer pan be cold when placed on the fire.

(iii.) Bring the water up to the boiling point, and maintain it at this for 3 or 4 minutes without removing the lid of the inner milk pan.

(iv.) Cool the milk down quickly by placing the inner pan in one or two changes of cold water without removing the lid.

(v.) When cooled down, aerate the milk by stirring well with a spoon.

Young now uses boiled milk with sanatogen added to it (2 grammes, *i.e.* 31 grains to the pint). He also permits the addition of cocoa, coffee and tea to the milk. Other points he notes are that cream added to soup or beef-tea improves the flavour and adds to the food value, that sound oysters are useful, that glucose added to beef tea (one teaspoonful to about 10 oz.) is valuable, and, quoting Harbin, that gelatin adds relish, lessens the nitrogenous waste and prevents hæmorrhage. Its food value, however, is nil.

Young also deals with the question of drinks, and describes the preparation of a very cooling beverage made from apples. He notes that ultramarine is found in sugar, and mica in barley, and therefore thinks that in preparing food and drinks it is wise to make a solution of the sugar first, allow this to settle for 6 hours and decant all except the bottom portion. The latter contains the ultramarine, which chiefly consists of silica, alumina and soda. In making barley water the barley must be well and frequently washed beforehand.

For the treatment of marasmus he speaks very highly of sanatogen, which is said to be a combination of pure casein and glycerophosphate of sodium. The large quantity of organic phosphorus is said to make it of value as a metabolic stimulant.

I have known a case in the Sudan in which milk was not well tolerated and sanatogen was used with success, but otherwise I do not know that typhoid cases require any special treatment, dietetic or otherwise, in a hot country, beyond such as may lessen the tendency to hyperpyrexia. Rogers believes this is best done by the cold pack treatment.

Ewart,[1] quoted by Young, advocates the Empty Bowel Treatment, or "plenty of food and no fæces." He gives peptonised milk, white of egg diffused in whey before peptonising, yellow of one egg a day, saccharin, lactose or a non-fermentable form of glucose, clarified honey, maltine, oil or cream, one ounce a day, common salt 10 to 15 grains to every half-pint of whey, watery extracts of vegetables, the juice of various fruits.

Young has modified this, giving the whey with cream and sanatogen. Of this prepared whey he administers 2½ to 4 pints in the 24 hours. It is easily prepared, easily digested, easily assimilated, and is declared to be a simple and perfect diet for the early stages of enteric fever.

Fæces. Nothing is more important in the diagnosis of disease in tropical countries than the examination of the fæces. This is specially true of the Sudan, a country in close proximity to and having much intercourse with Egypt, where, as is well known, metazoan parasites play no small part in the pathological field. There can be little doubt that in the

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[1] Ewart, W. (December 19th, 1905), "The Treatment of Typhoid Fever." *British Medical Journal*, p. 1720, Vol. II.

majority of cases an examination of the fæces should be a matter of routine procedure. Judging from the small amount of this class of work which falls to the share of the laboratories, these examinations are not frequently made in medical practice in Khartoum. Such work is disagreeable, especially in a hot country, and it has to be quickly conducted to be of value. Still I am very certain that a systematic examination of stools would well repay the time spent upon it, both as an aid to diagnosis and to amplify our knowledge of intestinal parasites and various bowel affections. **Fæces—** *continued*

Anyhow, some notes on the examination of fæces, a subject which has shared in the recent and general advance of medical knowledge, cannot fail to be useful.

Baumstark[1] points out that in order to properly test the capabilities of the intestine a special diet must be adhered to and it must fulfil certain requirements. These need not be tabulated here, for it is more to the point to quote from the notes dealing with the macroscopic, microscopic and chemical methods:—

The Macroscopic is the most important and determines the consistency, colour and smell of the fæces. The motion is thoroughly stirred with a wooden spatula and a quantity of the size of a walnut is put in a grater and ground down with a glass pestle, with a gradual addition of distilled water, to an absolutely fluid mass. When no more solid parts exist pour it on a large black plate. With normal intestinal action nothing but macroscopically recognisable remnants of cellular particles (rusk, gruel, cocoa) of the test diet should be found in the fæces. The following are of importance as pathological food remnants.

1. The remnants of the connective tissue and tendons from the minced meat which has been consumed; and these, owing to their light yellow colour, their fibrous form, and their firm consistency, can be recognised and most easily distinguished from mucus. Where any doubt exists, a small filament can be treated with a drop of acetic acid; in the case of connective tissue the filamentous structure vanishes; in the case of mucus it only then becomes visible. Quite isolated, small, sinewy filaments are to be found sometimes with quite normal digestions, but when in a great quantity they are always pathological.

2. Remnants of muscle which look like very small, brown-coloured splinters of wood. They are soft, become smaller when pressed, and disclose under the microscope muscle structure. In many cases connective tissue and remnants of muscle are to be met with in the same stool.

3. Remnants of potato, sago and similar transparent grain, which are frequently mistaken for mucus but which can be distinguished by their globular form and their hard consistency; they stand out above the level of the thin, spread out layer of the fæces. Under the microscope the potato cells appear to be either empty or filled with bluish (stained with iodine) coloured grains of starch.

The Microscopic Examination.—This is chiefly useful for the verification of the results obtained by the macroscopic examination; for example, in the differential diagnosis of connective tissue and mucus shreds. Three microscopic preparations are made, distilled water being added when the ground-up fæces are too hard. The first is simply a small particle placed on a slide and pressed by the cover glass into a thin layer. The second is rubbed up with a little drop of 30 per cent. acetic acid solution and held over a flame until it begins to boil; the third is rubbed up with a little drop of a strong solution of iodine in iodide of potassium (iodide 1, iodide of potassium 2, distilled water 50), and covered.

Under normal intestinal conditions the following should be observed in the preparations. 1. Preparation without addition—muscular fibres (flake-like formations coloured yellow and rounded at the edges with indications here and there of transverse striæ), some scattered small and larger yellow lime salts, light and dark yellow flakes consisting of sebates of lime, uncoloured (unstained) soaps, single potato cells empty, sparse remnants of chaff from gruel, and remnants of cocoa where cocoa was given instead of milk. 2. In the second preparation, when it is placed under the microscope whilst still hot, the larger lime salts and soap flakes are melted to neutral fat drops which, after they are cold, become solidified into small sebacic acid flakes. 3. In the third preparation, which is brown-coloured from the iodine, the potato cells now violet (but not blue) and sometimes violet-coloured sporules *(clostridium butyricum)* are met with.

Pathologically the following may be observed in the three preparations. In No. 1 broken pieces of muscle tissue in larger number with more clearly defined transverse striæ and sharp edges, neutral fat drops, sebacic acid, and soap needles in such quantity that they form the largest part of the preparation, and an abundant quantity of potato cells with more or less well-preserved grains of starch. In cold acetic acid preparations there are pathologically such a number of sebacic acid flocculi that all of the other component parts are in the minority. In the iodine preparation are bluish-coloured potato cells, as also scattered remnants of grains of starch, blue or violet sporules or bacterial flora, and oat cells which are yellow-coloured from the iodine.

One need not here detail the rather complicated chemical examination, but proceed to the author's consideration of the presence of mucus in the fæces:—

In many cases of sluggish motion without there being any symptoms of inflammation, a thin mucous coating of the fæces will be observed which causes the hard scybala to appear as if varnished. Many authorities do not regard this as arising from inflammation, but look upon it as ejected secretion. But where mucous shreds are seen constantly deposited externally on the scybala and also mixed with them during a lengthy period of observation, the existence of an inflammatory alteration of the mucous membrane may be inferred. The smaller the mucous particles are and the more they are mixed with the fæces so much higher is the part of the intestine from which they proceed. Their descent from the small intestine can only be assumed if the fæces are liquid and if the mucous flocculi are quite small and contain half-digested cells—that is to say, kernels of cells in their characteristic

[1] Baumstark, R. (June 16th, 1906), "Examination of the Fæces." *Lancet*, p. 1683, Vol. I.

Fæces—*continued*

arrangement. The larger the number of such cells the higher the degree of inflammation. The bilirubin colouring matter in the sublimate test and the presence of bilirubin crystals in the microscopical examination strengthen the assumption of the mucus originating from the small intestine.

The diagnostic conclusions to be drawn from the foregoing observations are as follows. The presence of connective-tissue remnants in the test-diet stools points with certainty to a disorder of the digestion, as only the gastric juice is able to dissolve raw connective tissue—*i.e.* from the centre and not from the well-cooked part of the meat. Achylia or hypochylia would usually be accountable for such a condition. Excessive peristalsis perhaps also plays an important part in some of these cases. Again, in excessive acidity associated with insufficient pepsine secretion (cases which not infrequently occur) the raw tissue meets with unfavourable conditions and appears again in the stools. A very minute examination of the stomach should be carried out in these circumstances. If muscle remnants are found macroscopically, or even in considerable amount microscopically, disturbance of the digestion in the small intestine may be inferred, for the stomach plays only an unimportant part in the dissolving of meat fibre and the digestion of meat does not take place in the colon. With both connective-tissue and muscle remnants in the stools it may be concluded that both stomach and small intestine are deranged.

With regard to the digestion of fat, it is to be observed that slighter variations in the fatty contents of the normal stool have no diagnostic meaning, for every stool contains fat in the normal fæces to the extent of 23 per cent. of the dry substance. A morbid increase of the fatty contents is proved at the macroscopic examination by the clayey quality of the light-coloured, nearly white fæces, by the very sour reaction, by the copious quantity of the fæces, and by their filmy appearance when rubbed up. Microscopical increase of the sebacic acid flocculi in the cold acetic acid preparation and neutral fat drops and salts of lime and soap crystals in a native preparation confirm the diagnosis. If the sublimate test show a complete absence of bile in the stools together with an increase of the fatty contents (in which case icterus also occurs), the cause of the disease is to be sought for in the biliary duct system. If, however, in conjunction with the increase of the fat contents (especially in the form of neutral fat drops in the native preparation), there are signs of muscle tissue simultaneously with the presence of hydrobilirubin and absence of mucus, then the pancreas is the seat of the disease.

In rare cases, where biliary deficiencies and pancreatic diseases are excluded, severe forms of intestinal disease, such as tuberculosis, amyloid disease, or tabes mesenterica, may be the cause of the derangement of fat absorption. The digestion of albumin is then likewise disturbed, and, as a rule, prior to the impairment of fat absorption. Moreover, purely functional disturbances of absorption limited to fats are sometimes observed. The simultaneous appearance, however, of mucus and processes of decomposition are decisive as regards an organic disease of the intestinal wall itself.

A complete absence of red or green colouring in the sublimate test shows absence of bile in the intestine (and icterus exists). If icterus be non-existent, then it a question of a temporary suspension of the secretion of bile. In the case of brown, fresh stools, absence of red colouring or a muddy colour would point to intestinal decomposition. When green colouring (bilirubin) is macroscopically demonstrated or if green colouring of separate particles—for example, of muscle tissue—is seen microscopically, it shows too rapid a passage through the colon; if the entire stool be coloured green, then most probably the small intestine is also involved. If there be a simultaneous appearance of small mucous flocculi described above, the cause is an inflammation of the mucous membrane of the small intestine.

Strasburger[1]* has a paper on much the same lines. He says that:—

Mucus can be recognised under the microscope, especially if one adds acetic acid. Mucus when recognised in the motion points to a catarrh in some portion of the intestine, and roughly the catarrh is in the large gut when the mucus is recognised macroscopically and in the small intestine when it is only detected microscopically. One learns more by noticing whether the mucus is intimately mixed with the fæcal material, and whether there are many cells associated with it. Blood and pus must be carefully looked for, and one must bear in mind that the red corpuscles may not be sufficiently preserved to be recognised under the microscope when the source is high up in the small intestine.

Bacteria and animal parasites are considered:—

Comparatively mild affections due to staphylococci can be readily distinguished by means of the microscope from the more dangerous infections due to streptococci. Typhoid, cholera, and dysentery bacilli can only be detected by culture, but tubercle bacilli may be demonstrated with comparative ease on cover-glass preparations. The amœbæ of dysentery, or certain forms of enteritis, can be seen under the microscope in fresh stools, but one must be careful not to press the cover-glass firmly on to the slide and also to keep the slide warm if one wishes to recognise their movements.

The latter injunction is unnecessary during the hot weather in the Sudan. At other times a warm stage or, what is far better, a Nuttall's microscope thermostat will be found necessary.

Daniels gives[2] a good account of the examination of fæces. The following may be quoted:—

Macroscopic examination. The points to observe are—

1. The presence or absence of blood, mucus, muco-pus or pus, and the arrangement relative to the stool of such a discharge.

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[1] Strasburger, J. (April, 1904), *Berl. Klin.* Quoted in *Medical Annual*, 1906.

[2] Daniels, C. W., "Laboratory Studies in Tropical Medicine." 2nd Edition, 1907.

* Article not consulted in the original.

Fæces—
continued

2. The colour of the stool and its consistence.
3. The presence or absence of evidence of gaseous fermentation.
4. The odour.
5. The reaction, determined as soon as possible after the stool is passed.
6. The bulk of the stools.
7. Any visible signs of animal parasites, such as the worms themselves or the proglottides or segments of tape-worms.

8. Mucus alone, or streaked or mixed with the blood, usually indicates inflammatory action in the lower bowel, not necessarily dysenteric. It may be caused by anything that sets up such inflammation, such as bilharzia, ulcerated hæmorrhoids, or chronic ulcerations of various kinds of the rectum. These latter include malignant growths, granulomatous growths, and the ulceration left as a sequela of dysentery. Rarely, mucus derived from the small intestines is passed with the fæces. Such mucus is recognised easily as it is stained with bile.

Clear mucus, whether streaked with bright blood or not, without any admixture of fæcal matter, is met with in early or acute dysenteric attacks. Turbid or purulent mucus, sometimes in large quantities and passed either without any stool or with solid-formed motions, is more indicative of a chronic ulceration of the rectum, from whatever cause.

Sometimes the mucus is passed in large masses and condensed, and may include much debris and numerous epithelial cells. In the condition known as membranous colitis, complete casts, several inches in length, of the rectum may be passed. These are usually twisted up when passed, and may be mistaken for worms. They can sometimes be floated out in water, and in any case the microscopic structure should render any mistake impossible.

With ulceration limited to the rectum, stools are often coated with mucus. The more intimately the mucus and blood are mixed with the fæces the higher up are the lesions from which the mucus or blood is derived. In some lesions the mucus is so intimately mixed with the fluid fæces that it is difficult to discern, but tilting the vessel from side to side will often indicate its presence by the manner in which the stool flows. In some cases it is better shown by adding water to the fæces, when the flakes or masses of mucus can be more readily seen, especially if the diluted fæces are poured from one vessel to another. Amœbæ are killed or have their motility destroyed by this addition of water, and therefore this method should only be adopted when the masses cannot be seen on inspection.

Blood may be passed, bright red or in clots, in large quantities. This is no proof that it is passed from the rectum, as if in sufficient quantity and not mixed with the fæcal contents of the intestine it need undergo very little change in passing through the large intestine. Such blood is occasionally passed in ankylostomiasis. If intimately mixed with the fæces it may have lost completely the red colour and appear black and tarry—melæna.

The consistence of the stool is of great importance, and it will be found that "looseness" of stools is of more importance in tropical practice than in England. In ulceration of the cæcum and upper part of the colon, even when this is acute and extensive, there need be neither visible mucus nor blood, nor even tenesmus. "Tropical diarrhœa" is frequently shown at post-mortem examinations to be dysenteric. It is very fatal. On the other hand, mucus and blood may be passed with formed or even hard stools when there are a few chronic ulcers high up in the large intestine.

In some forms of tropical diarrhœa, particularly that form known in the East as sprue, the stools passed are full of air bubbles and are undergoing active gaseous fermentation.

The odour varies so greatly with the diet that it is of minor importance. In the races subsisting mainly on a scanty vegetable diet the odour is singularly slight. The smell is mainly due to indol and skatol. In cases of dysentery associated with formation of sloughs the ordinary fæcal odour is replaced by the peculiar penetrating smell associated with that condition. Excessive decomposition of the stools may cause an increase in the intensity of the normal smell, or if the diet is mainly of carbohydrate food-stuff, no increase, but even a diminution.

Variations in the odour indicate changes in the decomposition of the contents of the intestine, often from variations in the food, but sometimes from variations in the "flora" of the intestinal contents, rarely from structural lesions of the intestinal wall.

The result of the administration of intestinal antiseptics is more often a diminution in the putrefactive changes in the contents of the bowel than any real improvement in the diseased condition of the intestinal wall.

The normal reaction of the fæces as determined by litmus is nearly neutral; when fasting it is acid, with a milk diet faintly alkaline. It is usually acid to phenolphthalein. In many cases of diarrhœa and dysentery this is replaced by a decidedly alkaline reaction. To determine the reaction the fæces must be examined as soon as they are passed, as a change rapidly occurs in most fæces, particularly when fluid, rendering them alkaline. Solid motions must be rubbed up with water in a mortar.

Indeed, the whole of this section is worth careful perusal. As regards parasites, the method of straining through muslin or fine wire gauze is described, while a point of practical importance in examining for ova in a watery stool is to remember that the eggs are heavier than the fluid and sink to the bottom of the vessel, from which they can be obtained by a pipette.

Sandwith,[1] under "Ankylostomiasis" *(page 9)*, notes that free moving larvæ in fresh fæces are never ankylostoma but are probably *Strongylus stercoralis*.

[1] Sandwith, F. M., "The Medical Diseases of Egypt," Part I., 1905.

Fevers. Under this heading one considers those obscure and indefinite febrile processes in the tropics to which so much attention has been recently directed, and on which, no doubt, a great deal of work still remains to be done.

In the first place, however, one may quote Sutherland's valuable paper[1] on the method of approaching a case of fever for the purpose of forming a diagnosis:—

The cause (he says) must be infective or non-infective. If infective, look for a parasite which must be either *(a)* Animal (amœba, piroplasma, Leishman-Donovan body, trypanosome, worm embryo or worm), or *(b)* Vegetable (coccus, bacillus, spirillum or fungus).

The infection must be either *(a)* of the general circulation, *(b)* of the spleen or lymph circulation, *(c)* a local infection of some mucous surface of entrance into, or exit from, the body, *(d)* an intoxication from without. Methods of procedure:—

1. Is it a general infection? *i.e.* of the circulating blood. If so, the parasite must be found in the circulation, or evidence must be obtained of its having been there.

Examine fresh blood at room temperature and on warm stage.

Examine blood films stained and with or without decoloration of the red cells.

A negative finding is inconclusive, for (1) the parasite may not be in the circulation at the time, *e.g.* the filaria embryo, or (2) the parasites may be few in number; and in any case one can hardly expect that a single drop of blood will always contain a parasite. Remember the value of drawing off a large quantity of blood, citrating it and incubating it at body temperature for some days. Sometimes parasites can only be found by making cultures from the blood or by making inoculations into susceptible animals.

If parasite not present, evidence of its previous presence shown, as in malaria, by broken up red cells and free pigment taken up by the leucocytes. The spirochæte of relapsing fever also leaves behind it in the blood, in the apyrexial periods, small coccus-like bodies.

(It is possible that in human spirochætal infections endoglobular bodies will yet be discovered similar to those found in the blood of fowls and geese.—A. B.)

Changes in the number of leucocytes, or in the differential count, are not distinctive. Taken with other signs, however, an increase of the small lymphocytes at the expense of the polymorphonuclears would point to Dengue fever. This point is not mentioned by Sutherland, but *vide* notes under "Blood" and "Dengue."

When the above methods fail to reveal a parasite or signs of its presence in the circulating blood, we are justified in assuming that the infection is not a septicæmia.

2. Is it a spleen or lymph infection?

Remember the circulating blood is inimical to microbes which take refuge in the lymph stream, and that the spleen, lymphoid tissues and lymph glands are the filtering media of the blood. If the parasite be in the spleen or in the lymph circulation its toxins must pass into the blood. Evidence of this is seen in leucocytosis or lymphocytosis. The latter is specially distinctive of spleen and lymph infections, particularly when the lymphocyte increase is of the large mononuclear variety.

In marked relative increase of large mononuclears, suspect malaria or kala-azar, but less marked increase occurs in enteric, tuberculosis and possibly syphilis.

When parasite is in spleen, look for spenomegaly.

Splenic puncture may be required, and reveals Leishman-Donovan bodies, malaria parasites, bacilli of anthrax, typhoid or micrococcus of Malta fever.

Presence of macrophages evidences splenic irritation. Absence of parasites and macrophages implies that there is no infection of the spleen.

As regards a parasite in the lymph circulation, consider the tonsil, vermiform appendix and superficial lymph glands. Look out for enlargement; or, in the case of the lymph sacs, effusion. Puncture of the tonsil or enlarged gland may reveal the parasite. Some infections, *i.e.* of Peyer's patches, deep lymph glands, etc., do not betray their presence, and are considered under local infection.

The parasite may be found in lymph sac effusions or, if these are sterile, the agglutination test may reveal the presence of tubercle, or a guinea pig may be inoculated.

[Here one may add that the ophthalmo-reaction would now be tried both for tubercle (Calmette) and typhoid (Chantemesse).—A. B.]

In all doubtful cases where the blood examination shows leucocytosis or lymphocytosis, where there are no physical signs and where spleen puncture is negative, lumbar puncture should be made to see if there be effusion into the meningeal lymph sac. Signs of exudation (as distinct from transudation) are positive, and the discovery of the parasite completes the diagnosis. When the exudate is sterile the case is likely to be one of tuberculosis, and confirmation is to be sought by the agglutination test and by inoculation into guinea pigs.

It is here that one may insert a note on the diagnosis of a condition not mentioned by Sutherland, and indeed only very recently brought to notice in India by Roberts and Bhandarkar, namely Acute Tuberculous Fever. This will be mentioned later on. Here we

[1] Sutherland, D., in "Enteric Fever in India, etc." Roberts, E., Calcutta.

need only indicate that at this stage, suspecting this condition, attention would be directed to the urine and search made for the tubercle bacillus in the manner to be presently described. **Fevers—** *continued*

Returning again to Sutherland's deductions, we find that

absence of signs of enlargement of lymphoid structures, of effusion into serous sacs, and negative results from lumbar puncture, imply that there is not a lymph infection, with the possible exception of infection of the visceral lymphoid tissues or of the deep lymph glands. The three infections most likely to occur in these situations are enteric fever, Malta fever and acute general tuberculosis, and further evidence can be gained as to these by the agglutination and sedimentation tests and by the diazo-reactions.

(Also possibly by the ophthalmo-reactions.—A. B.)

A positive diazo-reaction might imply either enteric fever or acute general tuberculosis, but positive agglutination and sedimentation reactions for enteric and Malta fevers distinctive, under proper conditions, of the technique employed.

3. Is it a local infection?

As a rule there are well-marked symptoms, but in deep-seated affections the physical signs may be overlooked. These secret local infections are generally associated with the micro-organisms of suppuration, with bacilli of the typhoid, colon or para-colon groups, or with the tubercle bacillus; and unless borne in mind they will often pass unrecognised. Septic throat infections are liable to be overlooked, and the resulting fever is often attributed to "malaria," simply because the throat was never examined.

The blood examination usually gives evidence of these infections, and a leucocytosis or relative increase of the lymphocytes or of the polymorphonuclears in the circulation, with absence of parasites in the peripheral blood, spleen or bowel, is always suggestive. A lymphocytosis points to tuberculosis or to a bowel infection by one of the typhoid or allied groups, and a polymorphonuclearcytosis to a local septic infection. The diazo-reaction may give evidence about the first two, the agglutination and sedimentation tests about the second, while the finding of peptone or albumose in the urine may indicate the third. A lymphocyte increase is of less value in diagnosing local infections than an increase of the polymorphonuclears, and calls for the diazo-reaction, the agglutination and sedimentation tests, and the search for tubercles in the chloride with the ophthalmoscope to clear up the issues. Increase of the polymorphonuclears, on the other hand, is distinctive, for it means local septic infection somewhere, and should send the observer back from the microscope to the patient to look more carefully for it. The conditions which should be examined for, one by one, are:—Oral sepsis, sore throat, appendicitis, abscess of the liver, subphrenic abscess, infective cholangitis, infective endocarditis, phlebitis, empyema, deep-seated pneumonia, abscess of lung, suppurative osteo-myelitis, suppurative periostitis, pyelitis, salpingitis, otitis media, mastoiditis, thrombosis of the lateral sinus, abscess of brain, cerebro-spinal meningitis, etc. With a polymorphonuclear increase in the blood, more thorough clinical examination will generally reveal the local mischief and settle the diagnosis, although the particular infecting microbe may not be determined until the necessary operation is performed.

If the diazo-reaction, the serum agglutination and sedimentation tests have given positive reactions and have indicated the nature of the infecting microbe, the diagnosis should be confirmed by microscopic examination of the sputum, fæces and urine, as the case may be, and cultures should be made and animals inoculated if necessary. The infecting micro-organism can generally be distinguished from others by making the agglutination and sedimentation reactions with the patient's blood, due regard being paid to the probability of a mixed infection (symbiosis).

Having isolated the infecting agent in the above ways, it should be cultivated, or kept in sealed tubes if the former be impossible, and subsequently used for prognostic purposes by testing, from time to time during the illness, the agglutinating power of the patient's blood and its bactericidal and phagocytic powers.

Apart from the side-room (laboratory) research, valuable information will be obtained from day to day at the bedside by watching the course of the disease, and the effects of treatment. The daily physical examination may reveal some local lesion which at first was not apparent; the time test may bring out distinctive rashes and show the affection to be one of the eruptive fevers, or may reveal characteristic features which identify the condition; the fever chart may become typical or the therapeutic quinine test abolish the fever. A consistently slow pulse will point to typhoid, the *tâche cérébrale* to typhoid or acute general tuberculosis, and so on through all the established diagnostic criteria.

If such a method of procedure be followed as a routine in doubtful cases, few will remain over to be returned as Simple Continued Fever, Remittent Fever, etc., terms which now fill the Returns. On the other hand, many slight cases of fever will occur where little or no information is got from the side-room examinations, and where recovery takes place before a scientific diagnosis has been made. In these cases it is hard to say what the fever should be called, and as they are generally associated with a little gastric, intestinal, bronchial, or uterine catarrh, it is best to return them under those heads. It would be well to regard every case of fever as having its origin and being in some infection or toxic process, *i.e.* in the reaction of the tissue cells thereto, and it is safer to conclude that heat and "chill" act only as predisposing factors.

In the above sketch, little has been said about toxic causes of fever as apart from infective causes, but doubtless toxic substances produced by intestinal and other parasites are capable of causing fever, and in most cases in India a search for ova in the stools, urine and sputum should be made. There is also the condition called "Fermentation fever," a subject to which Burdon-Sanderson was the first to direct attention, and which arises from the absorption of digestive ferments, fibrin ferment, extracts of fresh tissues, etc., and which is seen with gastro-intestinal disturbance, particularly in children, and after bruises, hæmorrhages, operations and the passage of a sound or catheter. It may also be noted that, as in the case of tuberculin, Professor Krehl has obtained a pyrogenetic albumose from cultivations of *B. coli commune*.

Fevers— *continued*

Mention has been made of the Acute Tuberculous Fever described by Roberts and Bhandarkar.[1] This is described as

> a continued fever of a remittent or intermittent type, lasting from a week to four weeks or more, and due to an acute invasion of the tubercle bacillus which during the febrile period and for a long time afterwards is found to be excreted in the urine. The main symptoms seem to be furred, but not "typhoid," tongue, skin dry and hot, no sweats, urine highly-coloured without albumen, and, specially characteristic, pain and tenderness in the epigastrium. The mind is clear, forming a marked contrast to the typhoid mental state. The liver is often enlarged, the spleen not so, and the bowels are usually constipated.
>
> Recovery may take place or the patient may drift into a tuberculous career, commencing with an indefinite kind of illness characterised by dyspepsia, fever relapses, depression and malaise. Some cases develop obvious tuberculosis. The diagnosis from liver abscess is very difficult, but is accomplished by finding tubercle bacilli in the urine by the following technique:—
>
> Centrifuge, wash the deposit once or twice in distilled water before fixation and use egg albumen in the fixing process. Stain by the Ziehl-carbol-fuchsine process with a final methylene blue (lightly done) as a counter stain. Other acid-fast bacilli have to be eliminated by the procedure recommended by Coles.
>
> Tests on guinea pigs which have been made were not completed at the time of publication.

These observations, which are very interesting, require confirmation, but, if this be forthcoming, a most valuable addition will have been made to our knowledge of obscure fevers, at least in India. Tuberculosis is far from being uncommon in the Sudan, and it will be well to keep on the look-out for cases answering to this description.

Rogers' book, already frequently quoted, is at the present time the standard work on tropical fevers, at least so far as India is concerned. The two chapters therein which bear especially on the subject under discussion are those respectively entitled "Unclassified Long Fevers" and "Unclassified Short Fevers." As regards the former, the author mentions Crombie's old classification and shows that his "remittent fever" is really typhoid, which was supposed to be uncommon amongst natives, while kala-azar accounts for another type. He then discusses certain doubtful, irregular, long fevers, dwelling principally on their incidence, and concludes that though they "may possibly belong to some one or more still undifferentiated tropical diseases, *yet they present no features incompatible with their being either paratyphoids*, including the class recently described by Castellani, or *early cases of sporadic kala-azar*, but they require further study and following up for long periods before their exact nature can be finally decided. Special reference is made to a Low Fever of European immigrants occurring in the damp, hot provinces of India, benefited markedly by change of climate or removal to a place with a dry soil, and due in all probability either to an enfeeblement of the heat-regulating mechanism or possibly to some undiscovered protozoal parasite. In the latter connection, he considers a leucocytozöon, but no parasites of this kind have been found in these cases. He also notes that a fever resembling kala-azar, but without the Leishman-Donovan bodies, has been described in the Philippines.

Mention is also made of undetermined fevers in China, such as the Double Continued Fever described by Manson and a similar form found by Rousseau amongst sailors at Hankow.

As regards the "Unclassified Short Fevers," we are now, thanks to Rogers' own work, on surer ground. He has described as a distinct entity, what he calls Seven Days' Fever, a condition which Sandwith believes exists in Egypt and which very probably occurs also in the Sudan:—

> The onset is sudden, in many cases rigor or chillness being present. Repeated rigors may occur resembling those of malaria. Rarely the onset is gradual.
>
> There is a flushed face, reddened conjunctivæ, often a listless, typhoid-like expression, while abdominal symptoms and even a few rose spots may suggest enteric fever.
>
> Pain in the back and limbs is common, but the joints are rarely affected, thus distinguishing the attack from dengue. Headache is common, severe and often frontal in site. The tongue is furred on the dorsum and has raw, red edges. It is like the tongue seen in influenza or dengue.
>
> Sickness may occur, but the bowels are usually regular. Abdominal pain and distention are not infrequent. Slight enlargement of the liver was found in 5 per cent. of cases, and of the spleen in only 7 per cent. Respiratory symptoms are absent. The pulse is like that in typhoid and paratyphoid, and is therefore somewhat characteristic.
>
> Rashes may occur but are not common. The typical temperature curve is of a saddle-back shape, the remission being slight, moderate or deep. Sometimes there is complete remission to normal before the terminal rise. There is also a type of case showing a continued typhoid-like curve and, as there is usually a terminal rise, cases coming late under observation are apt to be confusing.

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[1] Roberts, J. R., and Bhandarkar, R. S. P. R. (February 15th, 1908), "Preliminary Note on the Existence of an Acute Tuberculous Fever in India which has been confused with Continuous and Remittent Fevers." *British Medical Journal*, p. 377.

The name indicates the usual duration. From 4 to 8 days seems to be the limit. The convalescence is rapid and relapses do not seem to occur. **Fevers—** *continued*

The differential leucocyte count shows a considerable reduction in the percentage of the polynuclears with a corresponding increase in those of the lymphocytes and large mononuclears. The cause seems to be a bacillus which has been cultivated from the blood and resembles those of the coli group of organisms. It differs, however, from *B. typhosus* and *Paratyphosus*. The agglutination test was positive up to 1 in 40, but the reaction was not sufficiently constant to furnish a reliable diagnostic method.

Salicylate of soda and belladonna seemed to be the most useful drugs. Quinine only made the headache worse. No fatal case is reported.

The fever is much commoner in adult males than in females or children, and is much rarer in natives than in Europeans. The differential diagnosis is given, dengue being the great difficulty.

As regards distribution, it seems to occur principally on or near the sea-coast.

Clayton[1] studied an outbreak of Seven Days' Fever among the men of a cruiser temporarily quartered ashore in Bombay. The clinical symptoms are carefully described and suggestive facts brought forward to show that possibly the disease was transferred from sick to healthy through the medium of a suctorial insect. No organism was found in the blood, and experimental work was not conducted.

Another fever described is the Three Days' Fever of the hot season of Upper India, the cause of which is unknown but in which there is a large mononuclear increase. The temperature rises sharply and has a steady step-like decline, while headache and influenza-like pains seem to be the principal features. It is identical with the Chitral Fever described by McCarrison.[2]

I have myself suffered in Khartoum from what was either an attack of Seven Days' Fever or of Influenza. That it is was probably the latter is indicated by the marked benefit derived from a full and early dose of opium in the form of Batley's solution, but I am bound to say the rapid and complete convalescence after what was a short but severe and prostrating attack of pyrexia rather suggested this Seven Days' Fever, which, however, at that time had not been definitely distinguished.

There occurs also in Khartoum, more especially during the hot weather, a febrile condition of a few days' duration with indefinite symptoms of headache and malaise. In such cases I have been struck by the increase in the number of blood-plates which one finds when stained films are examined. Masses of platelets are visible usually along the edge or towards the ragged end of the film, though they also occur scattered through it. I am inclined to think there is some definite relation between this increase and the fever, but further observations are required, especially as regards the differential leucocyte count.

Castellani[3] has described cases of unclassed fever in Ceylon which, on superficial examination, may be taken for atypical forms of typhoid, paratyphoid, or malaria. The temperature is generally irregular; pulse frequently very slow; spleen not sensibly enlarged; no roseola; slight intestinal symptoms occasionally present; Widal test constantly negative; malarial parasites absent. The interesting bacteriological findings in these cases have already been discussed under "Bacteriology" (*page* 12).

Cropper[4] has described in Palestine a so-called Syrian Fever, which is generally quotidian or remittent in character, is accompanied by slight chills and more marked sweats, and is seen most severely in young infants who may have moderate enlargement of the spleen. In the red blood corpuscles the pale rods and melon-seed-shaped forms, known as Cropper's bodies, are found (*see pages* 111-112). In this fever, quinine is distinctly of use, but prolonged treatment is necessary.

Wellman[5]* has observed, in tropical Africa, a fever common in the dry season, of gradual evolution, long duration and characterised especially by persistent hyperpyrexia and a heavy mortality. In one case he found a great number of short bacilli in the blood.

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[1] Clayton, F. H. A. (June 15th, 1908), "A Contribution to the Study of Seven-day Fevers of the Indian Ports." *Journal of Tropical Medicine and Hygiene.*

[2] McCarrison, R. (January, 1906), "The Three Days' Fever of Chitral." *Indian Medical Gazette*, p. 7, Vol. XLI.

[3] Castellani, A. (January, 1907), "Notes on Cases of Fever frequently confounded with Typhoid and Malaria in the Tropics." *Journal of Hygiene*, p. 1, Vol. VII.

[4] Cropper, J. (February, 1907), "The Malarial Fevers of Palestine, and their Prevention." *Journal of the Royal Institute of Public Health*, Vol. XV., No. 2.

[5] Wellman, F. C. (1906), *Arch. für Schiffs. und Trop. Hyg.*, t. X.

* Article not consulted in the original.

Fevers— *continued*

An interesting account of Miliary Fever, or "Sweating Sickness," is given in the *Lancet* of November 17th, 1906, while Scholz[1]* has recently reported an epidemic of this curious disease, the infection of which Chantemesse suggests may be carried by the fleas of field mice. He noticed that the districts concerned had been overrun by these rodents and that many of the patients exhibited flea bites.

McCowen[2] has described very fully a Bilious Typhus Relapsing Fever, but as this really seems to be a special and definite form of true relapsing fever it will be considered under that heading.

Row[3] has a paper on serum reactions in obscure, irregular, continued fevers in India which led him to believe that both the *Bacillus enteritidis* of Gaertner and the *Bacillus coli communis*, especially the latter, stand in causal relationship to some of these forms of illness. Indeed, in some measure he anticipates the more recent work of Rogers and Castellani.

The same point was urged as regards Simple Continued Fever even earlier by Caldwell,[4] who quotes the still earlier work of Busch.[5]* At the same time, it must be remembered that in 1902 the agglutination reaction in all its phases was not so fully worked out as is now the case, and it seems desirable that definite evidence should be obtained as to the rôle of *B. coli* in these obscure but common cases.

Brief reference may be made to De Korté's paper[6] on Amaas or Kaffir Milk-pox, which seems to be small-pox mitigated by some undetermined factor or factors. It is not varioloid varicella, and is to be distinguished from what is known as Infectious Disease in Lascars. It is quite possible that amongst coloured races true modified small-pox occurs, and Colonel Hunter has told me that he has frequently wondered how often some of the outbreaks of so-called varicella in the Sudan are really mild and modified variola.

The point is one worthy of attention, albeit variola is steadily diminishing owing to general and efficient vaccination.

Filariasis. Low[7] has dealt with the unequal distribution of filariasis in the tropics. His researches were carried out in the West Indies, the distribution of *F. nocturna*, *F. demarquaii* and *F. perstans* being noted. He found that

where there was much clinical filarial disease, elephantiasis, etc., then the percentage of ordinary healthy people with embryos in their blood was high; where there was little disease, then the percentage was low. As regards *F. nocturna*, he found that its distribution in the various islands was very peculiar and interesting, and records his belief that there was something over and above the mere presence or absence of *C. fatigans* to account for the peculiarities he encountered. Much the same as regards distribution was true of *F. demarquaii* and *F. perstans*, even though, as he points out with regard to these parasites, we are not on such certain ground, as their proper intermediate hosts are unknown, unless the tick, *Ornithodoros moubata*, as Wellman believes, acts for the latter. Hence their irregular distribution may depend on the presence or absence of the intermediary. In a discussion on this paper, Sambon stated that he believed that several worms had been confounded under the name *Filaria bancrofti*. He suggested that hyper-parasitism might explain the absence of filariæ from certain regions. Leiper confirmed Low's statement as to the prevalence of *F. perstans* (*i.e.* the blunt-tailed embryos) in the blood of African natives in Uganda and E. Africa, and to the absence of the sharp-tailed embryos (*F. diurna* and *F. nocturna*). He pointed out, however, that though sharp-tailed embryos did not occur in Africa in man they were present in monkeys, and in these resembled very closely the embryos of *F. nocturna* found in the W. Indies. He also mentioned that distribution could not be determined on larval forms alone.

Sandwith stated that filariasis was not an extremely common disease in Lower Egypt, and that neither he nor anyone else in all probability could speak as to its incidence amongst Nubians. He also referred to Hayward's observations, who examined 400 patients in the hospital at Port Said and found that 15 per cent. of them were infected. Manson asked why it should be that in countries where *C. fatigans* was equally prevalent the disease it produced was very common in one, and in another it was very rare? He believed that if a satisfactory answer could be found, the means to counteract the pathogenic influence of the filaria would be also forthcoming. He further discussed the question of repeated re-infections and the remarkable fact that the propagation of the filaria was restricted in some way or other. He had obtained no evidence of hyper-parasitism and thought there must be some other explanation. He also discussed the relation of filariasis to elephantiasis and the fact mentioned by

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[1] Scholz. *Zeit. f. Klin. Med.* Vol. LIX., Nos. 5, 6.

[2] McCowen, W. T. (October, 1906), "Bilious Relapsing Fever." *Indian Medical Gazette*, p. 387, Vol. XLI.

[3] Row, R. (August, 1905), "Obscure, Irregular, Continued Fevers of the Typhoid Group, and their Probable Relation with different species of Bacilli of the Typho-Coli Race." *Indian Medical Gazette*, p. 292, Vol. XL.

[4] Caldwell, R. (February, 1904), "Simple Continued Fever: Its Cause and Prevention." *Journal of State Medicine*, p. 103, Vol. XII.

[5] Busch, F. C. (May 31st, 1902). *New York Medical Journal.*

[6] De Korté, W. E. (May 7th, 1904), "Amaas or Kaffir Milk-pox." *Lancet*, p. 1273, Vol. I.

[7] Low, G. C. (February 15th, 1908), "The Unequal Distribution of Filariasis in the Tropics." *Journal of Tropical Medicine and Hygiene*, p. 59, Vol. XI.

* Article not consulted in the original.

Low that in a country where filariasis was prevalent patients who were the subject of elephantiasis were rarely affected, or at all events seldom showed embryos of the parasite in their blood. This, he thought a strong proof that the parasite was the cause of the disease, something having happened to the subjects of elephantiasis previously, when they were actively infected with filariæ, which set up the elephantiasis and caused the death of the parasite. **Filariasis—** *continued*

In his reply, Low stated that though all elephantiasis was not filarial in origin the vast majority of tropical cases were due to this cause. He thought the death of the parent worm plus streptococcus might be the real factor. He had traced the development of elephantiasis in Barbados, where there was no malaria, from the initial fever and so-called ague, through recurrent attacks of lymphangitis to the true elephantiasis state. Embryos, however, were not found in the blood of those subject to the lymphangitis attacks, a curious fact which, however, did not vitiate his conclusions.

Wellman's suggestion, anticipated, however, in some measure by Feldman, has been mentioned. He[1] records work on *Ornithodoros moubata*, in which tick he found what he believed to be developmental forms of *F. perstans*. He thinks that the cycle is probably direct, from man to tick and from tick back to man. His experiments were carefully conducted and his results appear more reliable than those of Feldman,[2] who claimed that ticks (species not stated) take up *F. perstans* when sucking infected blood, that the worms undergo a certain development in the ticks and pass out with the eggs, being deposited in ripe bananas. These are ingested and the filaria bore their way into the tissues of the abdominal cavity and assume the adult form. Wellman was unable to confirm these observations and points out certain fallacies in them, one being that microscopic nematodes occur naturally in bananas.

Several recent papers deal with the development of filariæ in mosquitoes. Thus Lebredo[3] worked at the metamorphosis of filaria in the body of *Culex pipiens*. The paper goes minutely into details, and only portions need be quoted. Having traced the embryo from the blood to the stomach and then to the thorax of the mosquito, he finds that the embryo rests in the thorax and goes through the following transformations:—

(a) Narrowing and invagination of the tail.

(b) Invagination continues and the embryo grows shorter and wider.

(c) Widening and shortening continue and the invaginated portion forms a hyaline appendix.

(d) Period of growth and formation of the three lobes (at caudal end). He further states: "It happens sometimes, though rarely, that when the filaria reaches its maximum size, and starts on its way to the head, it may mistake the route, and wander towards the caudal extremity. The worm, however, will always keep in the fatty tissue, and close to the chitinous covering. These stray worms all proceed from the thorax. I have never met with a single embryo undergoing the process of metamorphosis in any other structure than the thoracic muscles."

The characters which lead one to the conclusion that the filaria has completed its cycle of development in the mosquito are stated to be:—

1. The arrival in the labium.
2. Complete development of the three caudal lobes.
3. Active motility.

Several other points are emphasised:—

(I.) When a mosquito falls into the water, if its cuticle be preserved, the filariæ it may contain are unable to escape, and perish by imbibition of water within a period of 24 hours. (II.) The filaria does not pass from the living mosquito in the act of suction of water or sugar. This rather tends to show that it is not likely to pass into bananas. Interesting experiments on the exit of the filaria from the proboscis are recorded. They lead to the conclusion that the heat in human blood is a factor in determining the exit of the filaria at the moment of the mosquito biting.

The author also concludes that only a series of accidents could bring the filaria to the human stomach as in the suggested imbibition of water.

The page on Histological Technique is certain to be found useful, and I transcribe it *in toto*:—

For studying the distribution of the filariæ in the body of the mosquito, sections are best used.

On the other hand, if we wish to follow the cycle of development of the parasite, of its dimensions and structure, we must dissect the infected mosquitoes.

Technique for the preparation of sections—

1. The live mosquito is placed in absolute alcohol. In this manner the penetration necessary for fixation and dehydration is obtained. Duration 24 hours.

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[1] Wellman, F. C. (July 20th, 1907), "Preliminary Note on Some Bodies Found in Ticks—Ornithodoros moubata—Fed on Blood containing Embryos of Filaria." *British Medical Journal*, p. 142.

[2] Feldman (April 15th, 1905), "On Filaria Perstans in the Bukota District." *Journal of Tropical Medicine*, p. 125, Vol. VIII.

[3] Lebredo, M. G. (May, 1905), "Metamorphosis of Filaria in the Body of the Mosquito *(Culex Pipiens)*." *Journal of Infectious Diseases*, Suppl., No. 1, p. 332.

Filariasis— *continued*

2. Removal of wings and legs in the same liquid, in a watch-glass.
3. Equal parts of absolute alcohol and ether, 24 hours.
4. Weak solution of celloidin, 24 hours.
5. Thick celloidin, 24 hours.
6. Mounting on block.
7. Chloroform, 20 minutes.
8. Eighty per cent. alcohol until ready to cut.

The sections were always stained in Böhmer's hæmatoxylin, followed in some cases by eosin and in others by van Gieson's solution.

Dissection—

The living insect is placed in a test-tube with a small amount of water, and is shaken until the wings become wet, and the insect floats on the water. The liquid is poured into a watch-glass, where the legs and wings are pulled off by means of forceps.

The further dissection should be made in the following order:—

Dissection of the abdomen upon a slide, constantly irrigating the mosquito with distilled water or weak salt solution. After dissection place under microscope and determine by medium power whether there are any filariæ. The head and thorax are transferred to another slide. Here the chitin is broken near the neck, and the head is transferred to another slide. Each part is thus dissected separately, and we are able to determine the localisation of the parasites. If the latter be well advanced in their development, they push their way out through the openings made in the chitinous covering at the time of dissection.

If we wish to preserve the specimens, the filariæ, if they are large enough, are transferred from place to place upon the slide, and repeatedly washed, while we clean and remove all detritus of the body of the mosquito from the slide. We finally add a drop of mixture of water, glycerin and formalin, and the preparation is covered and cemented. Such preparations may be studied with the immersion lens.

When the filariæ are too small to be isolated, it is best to leave them in the midst of the detritus, and to stain them as follows:—

1. Allow them to dry on the slide.
2. Fix with 95 per cent. alcohol, 1 to 2 minutes.
3. Place in water a few minutes.
4. Böhmer's hæmatoxylin, 1, 2 or 3 minutes, according to the intensity of the stain desired.
5. Wash in running water, 3 to 5 minutes.
6. Watery ·01 per cent. solution of eosin, ½ to 1 minute.
7. Wash in 95 per cent. alcohol.
8. Dry with paper.
9. Oil of cloves, 2 to 3 minutes.
10. Xylol.
11. Balsam.

The above technique may appear complicated, but it may be carried through in 8 to 10 minutes. Without staining, the filariæ, when they are too small or too few, may escape observation.

The dissection above described is not a careful dissection of the insect, but should be considered rather as a coarse fragmentation into sections as follows:—The stomach, intestine and ovaries in one piece; the thoracic muscle in four or more bundles; and the head and mouth parts. Care should be exercised not to lose a single fragment.

A somewhat similar paper is that by Ashburn and Craig[1] on the development of a human blood filaria *F. philippinensis* in *C. fatigans*. They summarise the changes as follows:—

In from 14 to 15 days the development is complete and the filaria has passed into the labium of the mosquito; the sheath of the embryo is lost in the stomach and the worm penetrates the stomach wall and reaches the muscles of the thorax where most of the developmental changes occur; during this period of time the filaria has increased in length from 0·32 millimeter to as much as 2·20 millimeters, and in breadth from 0·0065 to 0·02 millimeter; it has developed a well-marked intestinal canal, divided into œsophagus and intestine, a well-defined anus and three papillæ which are situated at the end of the tail; the mouth appears to be simply a circular cavity having no distinct lips. Development, so far as the morphology of the worm indicates, appears to be complete at about the eleventh day, the only changes occurring after that being a lengthening and narrowing of the filaria, which enables it to enter the labium of the mosquito. *F. philippinensis* does not seem to be pathogenic.

Fülleborn[2] has conducted an able research on the transmission of *Filaria immitis* of the dog by certain mosquitoes, notably *Anopheles maculipennis* and *Stegomyia calopus*. He has shown the important influence of humidity and temperature on the act of transference of

[1] Ashburn, P. M., and Craig, C. F. (March, 1907), "Observations upon *Filaria philippinensis* Development in the Mosquito." *Philippine Journal of Science*, p. 1, Vol. II.

[2] Fülleborn (1907), "Uebertragung von Filarienkrankheiten durch Mücken." *Arch. f. Sch. u., Trop. Hyg.*, t. XI., No. 20.

the filaria from the mosquito to the dog, and has devised ingenious experiments for his preliminary investigations. **Filariasis—** *continued*

Smith[1] has recorded from Sierra Leone the presence of two motile worms, apparently nematoids, and resembling developing filaria embryos, in the thorax of a mosquito, which appeared to be a non-blood sucker. This latter fact, however, required confirmation, and I can find no further reference to the subject.

Observations by Billet[2*] appear to show a more or less close genetic relationship between *F. loa* and *F. diurna*, while an interesting case of *F. loa* infection is described by Livon and Pénaud,[3*] of Marseilles, wherein the microfilaria were found in the blood, urine and saliva, and where there was a marked eosinophilia (55 to 70 per cent.).

Ward has studied *F. loa* in North America. He mentions all the cases on record and gives a very full and complete bibliography.

The geographical distribution of *F. loa* is given and the statement made that the West Coast of Africa is the proper home of the parasite. Strong recent evidence of the relation of the parasite to Calabar swellings is cited. Ziemann is quoted as finding the embryos in the swellings, but he believes that the latter are due to *F. perstans*, which he regards as the embryonic *F. loa*, while *F. diurna* (he says) does not differ from *F. bancrofti*.

Ward suggests that the swellings may be due to the discharge of waste materials of a toxic nature.

Several new facts have accumulated regarding filariasis in animals. These will be found detailed under the heading "Veterinary Diseases" *(page* 217*)*.

Nuttall[4] has published notes for the preservation of filaria intended to be sent for identification or study. These are as follows :—

EMBRYOS IN THE BLOOD

1. Drop blood in a solution of 1 in 4000 perchloride of mercury in 8 per cent. saline. Mix by shaking, cork and send without more ado.

2. Drop blood into solution of iodine added to 8 per cent. saline, in quantity sufficient to give the saline a pale sherry colour. Mix, cork and send.

3. Drop blood into 10 per cent. boiling formalin solution, allow sedimentation to take place, then decant and preserve in 5 per cent. formalin.

4. Make blood smears on cover glasses, dry, pack them back to back, surround with thin paper (paraffined better), put into small box into which paraffin, melting at not too high a temperature, say 50° to 60° C., has been poured. Now pour in fresh melted paraffin so that the cover glasses lie in a block of paraffin.

Note.—I have devised this method in the hope that it will work with films collected in the tropics, excluding their growing mouldy and altering with regard to stains.

ADULT FILARIÆ

1. Place worms in saturated perchloride of mercury for 24 hours, then rinse with water for 30 minutes and place in 70 per cent. spirit to which a little iodine has been added, so as to give it a sherry colour. Cork and send.

2. Place worms in dish, pour on boiling saturated perchloride, rinse as before and store in iodinised 70 per cent. alcohol.

3. Drop worms direct into 4 per cent. formalin in 8 per cent. saline ; cork and send.

The different methods can be used according to the convenience of the worker.

Low[5] gives the following method for making permanent preparations of filarial embryos. Spread a large drop of blood as a film on a slide and allow to dry. Place in water till all the red colour of the blood disappears and then apply a few drops of fuchsin, methylene blue or hæmatoxylin for a few minutes. Wash again in water, dry and mount in Canada balsam.

O'Brien[6] mentions two cases in which atoxyl, given intravenously, caused the embryos of *F. nocturna* to disappear from the blood. He administered from 5 to 40 minims of a 5 per cent. solution, continuing the treatment in one instance for about two months.

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[1] Smith, F. (May 1st, 1905), "The Development of Filariæ in the Mosquito." *Journal of Tropical Medicine*, p. 140, Vol. VIII.

[2] Billet, A., *C. R. Soc. Biol.*, t. LXI., p. 507.

[3] Livon, J., and Pénaud, December 1st, 1906, *C. R. Soc. Biol.*

[4] Nuttall, G. H. F. (October, 1904), "Directions for Preserving Filariæ." *Indian Medical Gazette*, p. 391, Vol. XXXIX.

[5] Low, G. C. (February 16th, 1903), "Method of Mounting Specimens of Bilharzia Eggs, Filarial Embryos, and Small Worms of Any Kind." *Journal of Tropical Medicine*, p. 67, Vol. V.

[6] O'Brien, R. A. (May 15th, 1908), "The Administration of Atoxyl Endovenously in Filariasis." *Journal of Tropical Medicine and Hygiene.*

* Article not consulted in the original.

Filariasis— *continued*

Personally I cannot say much about filariasis in the Sudan. The only filaria I have found in human blood in this country was *F. perstans*, and that in a Ugandese at Taufikia. In the only case of elephantiasis whose blood I examined at night I failed to find embryos. Neither have I found them in the blood of monkeys *(Cercopithecus)*. Elephantiasis does occur, but is apparently not a common disease. Singer[1] records that in South-Western Abyssinia elephantiasis occurs, but is rare. Ensor has recently reported filariasis to be common in the southern Bahr-El-Ghazal Province of the Sudan.

Culex fatigans is common all over the Northern Sudan and occurs also in large numbers on the Upper White Nile, but in these southern territories *Mansonia uniformis* is more likely to play the part of a filaria vector. A great deal of useful information could be obtained by the systematic examination of bloods at night in the various hospitals. So far as I know nothing has been done in this direction. It would entail hard work in such a trying climate, but during the winter months the examinations could be made without very much discomfort or fatigue. It would seem advisable, in the case of elephantiasis patients brought in for operation, to enforce the use of a mosquito net at night, as where *C. fatigans* or *M. uniformis* are present the disease might be transferred from the sick to the healthy.

The question of infection with *Filaria medinensis* is considered under the heading "Guinea Worm" *(page 83)*.

Filters. An important practical paper of recent date is that by Bulloch, Craw and Atkin[2] on the relative efficacy of the Doulton, Berkefeld and Brownlow filters. An investigation such as they conducted was urgently required, for doubt had been thrown on the efficacy of the Berkefeld filter, despite the statements of Woodhead and Cartwright Wood. The later investigators conclude that—

> Of all the filters tested, the Doulton filters alone uniformly prevented the direct transmission of micro-organisms; the Berkefeld filters all permitted direct transmission, with one exception, and all the Slack and Brownlow filters gave contaminated filtrates immediately. In the case of the Berkefeld filters and the Slack and Brownlow filters the germs passed through within 15 minutes from the commencement of filtration, whereas seven out of ten Doulton filters withheld the organisms and gave sterile filtrates for four days and in three cases for longer periods. As these tests were made with a water pressure varying from zero to 32·5 lb. per square inch, often in a few seconds, the above examination appears to us to be not only severe but also conclusive.

Craw has a supplementary paper on the grain of filters and the growth of bacteria through them, and brings out some interesting points. He finds that the size of pore is very small in the Doulton filter, relatively greater in the Pasteur-Chamberland, very much greater in the Berkefeld, and in the case of the Slack and Brownlow filter the pores are of striking magnitude. These points are very well brought out in a series of photographs.

He concludes that the grain of a filter is a very important, if not the most important, factor in governing the growth of bacteria through the filter mass, and that in the filtration of fluids containing colloidal substances, suspended matter or micro-organisms, the chemical nature of the filter mass will be eliminated very rapidly as a factor in the efficiency of the filter owing to the formation of a coating of foreign material, derived from the fluid, over the chemically active surface.

As regards water filtration on a large scale, much valuable information will be found in Hazen's work,[3] which is far too little known on the eastern side of the Atlantic. A short *résumé* of the subject, comparing the English sand filter with the American mechanical filter, is given by Robinson.[4] As he points out, the latter, where a chemical precipitant is employed, are specially applicable to very turbid and muddy waters. The efficiency of the mechanical filters depends entirely on the use of coagulants, hence the value of a paper by Whipple and Longley[5] on the necessity of using basic and not neutral alum in mechanical filtration. With neutral alum the bacterial efficiency was only 95·5 per cent., while with basic alum 98·4 per cent. was obtained. As a result they contend that specifications for filter alum should always require that there shall be a substantial excess of alumina, that is,

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[1] Singer, C. (January 16th, 1905), "Notes on Cases met with in South-Western Abyssinia in March, 1904." *Journal of Tropical Medicine*, p. 17, Vol. VIII.

[2] Bulloch, W., Craw, J. A., and Atkin, E. G. (January, 1908), "On the Relative Efficacy of the Doulton, Berkefeld and Brownlow Filters." *Journal of Hygiene*, p. 63, Vol. VIII.

[3] Hazen, Allen, "The Filtration of Public Water Supplies." New York, 1903.

[4] Robinson, W. J. (September 15th, 1906), "Artificial Purification of Water Supplies." *British Medical Journal*, p. 623, Vol. II.

[5] Whipple, G. C., and Longley, F. F. (February, 1906), "Experience with the Use of a Non-basic Alum in Connection with Mechanical Filtration." *Journal of Infectious Diseases*, Suppl., p. 166, Vol. III.

that the alum shall be distinctly basic. This is usually so in any case when the available alumina exceeds 17·5 per cent. The following is the type of specification recommended :— Filters—cont

The basic sulphate of alumina shall be guaranteed to contain 17 per cent. of alumina (Al_2O_3), soluble in water, and of this amount at least 5 per cent. shall be in excess of the amount theoretically necessary to combine with the sulphuric acid present. It shall not contain more than 0·1 per cent. of matter insoluble in water, and it shall be practically free of chips and débris of all kinds. It shall not contain more than 0·5 per cent. of iron (Fe_2O_3), and the iron shall be preferably in the ferrous state. The alum shall be crushed to pieces of such a size that they will pass through a ring three inches in diameter.

If bidders so desire, they may bid on a product containing a large amount of soluble alumina, stating, however, the amount which they guarantee. The additional strength will be taken into account in deciding where to place the order.

Mention may be made of closed Candy filters, specially adapted for waters containing iron ; and the Bell filters, which seem to be an adaptation of the American mechanical filter, are closed, appear to be efficient bacteriologically, are easily cleaned, and are likely to have a future before them as they are not very expensive.

Smith[1] deals with modern methods of water filtration, considering first the sand filter used both in the old manner and along with the "Howard" process, which consists in running on to the surface of the filter a strong solution of aluminium sulphate at the beginning of every filtering period, thus inducing a rapid formation of the so-called vital layer. He also notes the spreading of the raw water over the filter bed by means of a revolving sprinkler—a recent innovation. Secondly, he describes mechanical filters, distinguishing four chief types—the Jewell, the Bell, the patent open and pressure filters of Reeves, containing quartz crystals, and the Candy filter. A warning is issued against the use of any of these types without due precautions as to skilled oversight, etc. It is advisable that such filters should be tested with the water which it is proposed to purify.

As regards the question of domestic and public filters in Khartoum, *see* "Sanitary Notes" (Third Report).

Flies. Under this heading a few papers will be noticed dealing not only with biting flies but with those flies which from their filthy feeding habits may carry disease germs.

Austen[2] deals with the house-fly and certain allied species as disseminators of enteric fever among troops in the field. He quotes Major Smith, who says :—

an old idea of some Anglo-Indian surgeons was that dysentery could be caught by using the same latrine as a dysentery patient. There may be something in this. Experiments on animals have shown that the disease can be inoculated per rectum. The ubiquitous fly may, therefore, be a dysentery incubator in open camp latrines.

Enteric fever may possibly be conveyed in a similar manner, and Austen observes that many cases of intestinal myiasis due to the larvæ of flies belonging to the genus Homalomyia (*H. canicularis*, Linn., and *Scalaris*, Fabr.), are probably to be traced to the parent flies having oviposited on the anus when the patient has been using some country privy where these flies are common. He then proceeds to describe the characteristics, life-history and general bionomics of the commonest latrine-haunting flies, especially as regards South Africa. He notes that *Homalomyia canicularis* is very often mistaken for *Musca domestica*, the true house-fly, but it is smaller, narrower, and the fourth vein runs straight to the tip of the wing instead of being bent up at an angle as in *Musca*. It is often called a "young house-fly" by those who do not know that the larval stage is the period of growth in insects and that no insect grows, in the true sense of the word, after attaining the perfect state.

After dealing in detail with the house-fly, Austen mentions two other filth carriers, the plum-coloured or metallic-green or bluish-green *Pycnosoma* flies, *P. marginale* and *P. chlorophyga*, which also breed in decaying animal and excrementitious matter and accordingly haunt latrines.

Methods of preventing the access of flies to latrine trenches are given and a suggestion made, that light metal covers, provided at regular intervals with apertures, closed by spring flaps which could be actuated by the feet of the users, might prove useful, especially if combined with the discharge into the trench of some such fluid as kerosine or crude creolin in solution.

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[1] Smith, W. R. (May, 1908), "Modern Methods of Water Filtration." *Journal of the Royal Institute of Public Health*, Vol. XVI., No. 5.

[2] Austen, E. E. (June, 1904), "The House-Fly and Certain Allied Species as Disseminators of Enteric Fever among Troops in the Field." *Journal of the Royal Army Medical Corps*, p. 651.

Flies— *continued*

A very excellent paper on House-Flies is that by Howard.[1] He deals with all the flies which invade houses in the United States, mentioning *Musca domestica*, *Stomoxys calcitrans*, *Pollenia rudis* (the cluster fly), *Muscina stabulans*, a stable fly exactly like the common house-fly, and three species of so-called blue-bottle fly, namely, *Calliphora erythrocephala*, the blow-fly, the small blue-bottle fly, *Phormia terrænovæ*, and the green *Lucilia cæsar*.

Homalomyia canicularis and *Homalomyia brevis* also receive attention, as does the small, jet-black window fly, *Scenopinus fenestralis*, which breeds in the dust under carpets. The small and slender *Sepsis violacea*, often seen on window-panes, is also mentioned and figured.

The chief natural enemies of the house-fly are detailed. These are the house centipede, a small reddish mite and, in its larval stage, hymenopterous parasites and predatory beetles. *Empusina muscæ*, however, a form of fungus, is its principal destroyer, but its action is more than counterbalanced by the rapidity of development of the fly.

Remedial and preventive measures as regards the breeding of house-flies are considered. Stable manure is their special breeding-place, and experiments were tried with various substances. It was found that by spraying eight quarts of fresh horse manure with one pint of kerosene and afterwards washing down with one quart of water the manure was thoroughly rid of living maggots. While good results were obtained on a small scale, this method employed on an economical basis proved inadequate when applied to large quantities of manure. Chloride of lime also failed on a large scale and the best results were obtained by the preparation of a special receptacle for manure. A chamber 6 feet by 8 feet was constructed with a door opening into the stable, and a window with a wire screen. In the outside wall of this building another door was placed. All manure was thrown into this chamber and each morning a shovelful of chloride of lime was scattered over the fresh layer. After ten days or two weeks the manure was removed viâ the outside door.

House-flies also breed in human excrement and, as recently discovered, in cow-dung. Howard mentions a French method for keeping them away from privies and cesspools. Residuum oil is used, two litres per superficial metre of the pit. This is mixed with water, stirred with a stick, and thrown into the receptacle. Not only does this kill larvæ and prevent the entrance of flies, but it practically turns the cesspool into an anærobic tank.

Finally, Howard quotes certain U. S. regulations which might well be adopted with appropriate modifications in all towns:—

> All stalls in which animals are kept shall have the surface of the ground covered with a water-tight floor. Every person occupying a building where domestic animals are kept shall maintain, in connection therewith, a bin or pit for the reception of manure, and, pending the removal from the premises of the manure from the animal or animals, shall place such manure in said bin or pit. This bin shall be so constructed as to exclude rain-water, and shall in other respects be water-tight, except as it may be connected with the public sewer. It shall be provided with a suitable cover and constructed so as to prevent the ingress and egress of flies. No person owning a stable shall keep any manure or permit any manure to be kept in or upon any portion of the premises other than the bin or pit described, nor shall he allow any such bin or pit to be over-filled or needlessly uncovered. Horse manure may be kept tightly rammed into well-covered barrels for the purpose of removal in such barrels. Every person keeping manure in any of the more densely populated parts of the district shall cause all such manure to be removed from the premises at least twice every week between June 1st and October 31st and at least once every week between November 1st and May 31st of the following year. No person shall remove or transport any manure over any public highway in any of the more densely populated parts of the district except in a tight vehicle which, if not enclosed, must be effectually covered with canvas, so as to prevent the manure from being dropped. No person shall deposit manure removed from the bins or pits within any of the more densely populated parts of the district without a permit from the health officer. Any person violating any of the provisions shall, upon conviction thereof, be punished by a fine of not more than 40 dollars for each offence.

Fresh proof of flies as carriers of infection is furnished by the work of Buchanan[2] with *M. domestica* and the blue-bottle fly *M. vomitoria*. He describes the form and structure of the fly's tarsus and shows how well adapted it is for carrying a large amount of infective material. He had positive results as regards transference of specific germs and growth on media, with swine fever, staphylococcal abscess, pulmonary tuberculosis and anthrax. It was less easy to prove conveyance of infection in the case of enteric fever, but this also was successfully accomplished, the culture medium employed being that of MacConkey as modified by Grünbaum and Hume.

Another useful paper is that by Hewitt.[3] He believes that *Homalomyia canicularis* is quite as guilty as *M. domestica* in the dissemination of infectious disease. He notes that in

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[1] Howard, L. O. (September 21st, 1906), United States Dept. of Agriculture, Bureau of Entomology Cir., No. 71. Revised Edition.

[2] Buchanan, R. M. (July 27th, 1907), "The Carriage of Infection by Flies." *Lancet*, p. 216, Vol. II.

[3] Hewitt, C. G. (December 11th, 1907), "On the Bionomics of certain Calyptrate Muscidæ and their Economic Significance, with Especial Reference to Flies Inhabiting Houses." *Journal of Economic Biology*, p. 79, Vol. II.

England the method to bring about its decrease would be the universal adoption of the water-closet system and also the proper disposal of kitchen refuse. Flies—*continued*

Newstead[1] has studied very fully the habits, life-cycle and breeding-places of the common house-fly in Liverpool. The paper must be consulted for details but the conclusions regarding breeding-places and the preventive measures recommended may be tabulated here.

i. The chief breeding-places of the house-fly are:—

(a) Stable middens containing fermenting manure or a mixture of this and cow-dung;

(b) Middens containing fermenting, spent hops; and

(c) Ashpits containing fermenting vegetable matter, or about 25 per cent. of the total number of pits examined.

ii. That covered ashpits and middens were as badly infested as those which were open.

iii. That house-flies breed in all temporary collections of fermenting matter.

iv. That house-flies breed in relatively small numbers in ashpits where no fermentation takes place.

v. That they do not breed in ashpits which are emptied at short intervals, or in the patent bins.

vi. That the use of disinfectants in ashpits does not prevent the flies breeding in such receptacles.

vii. That very dry or excessively wet ashes or moist cow-dung does not harbour them.

viii. That the presence of fowls (not ducks or geese), which had free access to the stable middens, reduced the number of larvæ and pupæ to a very marked extent.

ix. That the life-cycle of the fly, in all kinds of fermenting materials, is reduced to the minimum period of ten to 14 days; and that in the absence of such artificial heat the cycle may occupy a period of from three to five weeks or more, according to the temperature of the outside air.

x. That the house-flies do not depend entirely upon excessively warm weather for breeding purposes, though in hot seasons they would breed much more rapidly in non-fermenting materials, and their numbers, under such conditions, would be greatly increased.

If house-flies are to be reduced to a minimum, I would submit the following suggestions . . . . .

1. That stable manure and spent hops should not be allowed to accumulate in the middensteads during the months of May to October inclusive, for a period of more than seven days.

2. All middensteads should be thoroughly emptied and carefully swept at the period stated in 1.

The present system of partly emptying such receptacles should in all cases be discontinued.

The walls of middensteads should also be cemented over, or, failing this, the brickwork should be sound and well pointed.

3. That all ashpits should be emptied, during the summer months, at intervals of not more than ten days.

4. That the most strenuous efforts should be made to prevent children defæcating in the courts and passages; or that the parents should be compelled to remove such matter immediately; and defæcation in stable middens should be strictly forbidden. The danger lies in the overwhelming attraction which such fæcal matter has for house-flies, which latter may afterwards come into direct contact with man or his foodstuffs. They may, as Vedeer puts it, "in a very few minutes . . . . . load themselves with dejections from a typhoid or dysenteric patient not as yet sick enough to be in hospital or under observation, and carry the poison so taken up into the very midst of the food and water for use at the next meal. There is no long, roundabout process involved."

5. Ashpit refuse, which in any way tends to fermentation, such as bedding, straw, old rags, paper, waste vegetables, dirty bedding from the "hutches" of pet animals, etc., should, if possible, be disposed of by tenants, preferably by incineration, or be placed in a separate receptacle so that no fermentation could take place. If such precautions were adopted by householders, relatively few house-flies would breed in the ashpits, and the present system of emptying such places at longer intervals than, say, four to six weeks, might be continued.

6. The application of Paris Green (poison) at the rate of 2 oz. to one gallon of water to either stable manure or ashpit refuse will destroy 99 per cent. of the larvæ. Possibly a smaller percentage of Paris Green might be employed with equally good results.

One per cent. of crude atoxyl in water kills 100 per cent. of fly larvæ.

The application of either of these substances might, however, lead to serious complications, and it is very doubtful whether they could be employed with safety. Paris Green, at a rate of 1 to 2 oz. to 20 gallons of water, is used largely as an insecticide for fruit pests. It does no harm to vegetation when applied in small quantities; but cattle might be tempted to eat the dirty straw in manure which had been treated with this substance, and the results might prove fatal if large quantities were eaten.

7. The use of sun-blinds in all shops containing food which attracts flies would, in my opinion, largely reduce the number of flies in such places during hot weather. Small fruiterers' and confectioners' shops, as a rule, are not shaded by sun-blinds, and in their absence flies literally swarm on the articles exposed for sale.

8. The screening of middensteads with fine wire gauze would, undoubtedly, prevent flies from gaining access to manure, etc., but it is very doubtful if this method would meet with any marked success. The gauze would rapidly oxidize, the framework supporting it would probably warp, and numbers of flies would be admitted whenever the receptacle was opened. Moreover, the erection of such a structure would prove a great inconvenience and a hindrance to the removal of the refuse. This, however, does not prejudice the possibility of erecting a good fly-proof screen in the future.

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[1] Newstead, R. (February 29th, 1908), "On the Habits, Life-Cycle and Breeding-Places of the Common House-Fly (*Musca domestica*, Linn.)." *Annals of Tropical Medicine and Parasitology*, p. 507. Vol. I., No. 4.

Notes are added on *Calliphora erythrocephala*, the "Blow-fly." It feeds on the fæces of man to a greater extent than the house-fly, and is very partial to fruits of various kinds, hence it is very likely to be a vector of disease.

Franklin[1] writes with Indian experience. He thinks that there the house-fly breeds in great numbers in night-soil and not in the vicinity of cook-houses, which apparently only constitute its feeding grounds. He has made observations on these terrible pests, owl midges, which, if confirmed, are likely to be helpful, for he finds that these so-called "moth-flies" (in Khartoum they are always, though erroneously, termed sand-flies), breed out in material taken from the neighbourhood of cook-houses, for example, what is found in the drain at the back of cook-houses. He describes the larvæ and pupæ, and imagines from some experiments, it would seem, that these tiny diptera can, like the house-fly, carry about excrementitious material.

Another Indian paper is that by Major F. Smith, who draws attention to a very important matter as regards the tropics, namely, the breeding out of *M. entæniata* and *Anthomyia tonitrui* in scattered deposits of human fæces, and the occurrence of maggots of these flies *in the soil under the fæcal masses*. As the fæces dry and crumble, the maggots bury themselves in the earth, finding a passage by way of cracks or the holes made by worms or dung-beetles. They were found to take about 14 days to hatch out.

Dog excrement was also found to serve as a breeding-place for *M. entæniata*, which, be it noted, haunts houses and hospital wards. Cow-dung and earth under it harboured maggots of *M. domestica* and *M. entæniata*, while horse and donkey dung yielded larvæ of *M. domestica*, but did not seem to be a very favourite breeding-ground, at least in dry weather.

Experiments seemed to show that house-flies did not breed in ordinary ground as distinguished from organic deposits.

The question of flies and their breeding-places, as regards Khartoum, will be discussed under "Sanitary Notes." One need only note here that Major Smith's observations apply to the Sudan as well as to India. They have been amply confirmed by Mr. King.

Passing now to biting-flies, we find a reference to the work of Legaillon[2] on the life-history of *Tabanus quatuornotatus*. This is of special interest in view of King's work on *T. biguttatus* in the Sudan.

The eggs are laid in bunches in places that may be either dry or moist, for instance, on the leaves of plants, and are at first white, but soon darken. As is already known, some larvæ may be aquatic and others terrestrial, while their habits are carnivorous. The eggs of the species under consideration hatched out in 14 days (in June), and are white and transparent. They feed on dead prey and on organic detritus, and perhaps even on sluggish, living animals. They can live under varied conditions of moisture and even in water.

One can only give some references to other papers or works on biting diptera. Thus there is Austen's beautifully illustrated work on the British Blood-sucking Flies, while Bouvier has a good paper in the *Annales de l'Institut Pasteur* of 25th July, 1906. Dudgeon deals with the biting-flies of the West Coast and the Niger in the *Journal of Tropical Medicine* for 1st November, 1906, and 15th November, 1906, and Newstead with those of the Congo Free State in the *Annals of Tropical Medicine and Parasitology* of 1st February, 1907. (Series T.M., Vol. I., No. 1).

A German work by Grünberg is entitled "Die Blutsaugenden Diptera," while a French paper by Surcouf, with coloured plates, occurs in the *Archives de Parasitologie* for 1st August, 1907. (Tome XI., No. 3)

Picard[3]* gives the distribution of the following species of *Stomoxys* which are found in Tropical Africa: *S. calcitrans*, L.; *S. Korogwensis*, Grünberg; *S. bouffardi*, Picard; *S. inornata*, Grünberg; *S. glauca*, Grünberg.

[Those interested in the distribution of Tabanidæ will find a list of those which occur in Angola given by Wellman in the *Journal of Tropical Medicine and Hygiene* for April 15th, 1908, p. 117, while Austen deals with new African Tabanidæ in the *Annals and Magazine of Natural History*, for March, 1908. In the same journal Miss Ricardo commences a descriptive paper on 30 new species from Africa and Madagascar, which is continued and completed in the April number.]

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[1] Franklin, G. D. (September, 1906), "Some Observations on the Breeding-Ground of the Common House-Fly, and a Description of a Species of Moth-Fly." *Indian Medical Gazette*, p. 349, Vol. XLI.

[2] Legaillon, A. (February 15th, 1906), "Egg Deposition and the Larval Life of the Tabanidæ." *Journal of Tropical Medicine*, p. 58, Vol. IX.

[3] Picard, F. (1907), "Sur quelques Stomoxys de l'Afrique occidentale." *Bull. Soc. Entomol. de France*, No. 18, p. 312.

* Article not consulted in the original.

Food. It is quite impossible to deal in any detail with the mass of recent papers on this subject. We propose only to mention a few which refer to food in the Tropics or to tinned foods or to foods suitable for travel, as being most likely to be useful from a Sudanese standpoint.

Simpson[1] points out that the defect likely to be in the diet of a European in the Tropics is that it is too nitrogenous and fatty. This is due to the tendency to continue a diet suitable for a temperate climate. The result is that too much meat is taken, which places a strain on the excretory organs and causes disorder of the digestive functions. The condition which ensues predisposes to sunstroke. Animal food should be diminished and a more liberal allowance of vegetable food taken. The latter supplies the necessary constituents in a less stimulating form and one more suited to a climate in which congestion of the abdominal viscera is specially apt to occur. In the Tropics, where the temperature is nearly as high and sometimes higher than that of the body temperature, there is not the same necessity for fatty food as in colder climates, and the substitution of fruits and farinaceous substances for oleaginous articles will not only be more grateful to the taste but will prevent intestinal disorders which, under the circumstances, are likely to be produced by a diet which is too fatty. It would be a mistake, however, to exclude meat altogether from the diet, and, in the case of special hard work out-doors, meat may be increased in the diet with advantage. Carbohydrates furnish energy with a moderate production of internal heat, hence they are very valuable in the Tropics, especially indigenous cereals such as maize, rice and the native lentils, as they contain less nitrogen. Sugar is excellent when energy is to be liberated rapidly with the least tax upon the digestive system. Simpson cites the case of the debilitated cavalry horses in the Philippines which recovered health and energy when their coarse grass or hay was sprinkled with molasses or sweetened water. He also refers to the deficient nitrogen in the diet of the Hindoo, rice itself, though agreeable and digestible being deficient in nourishment. In grain-eating populations starchy constituents bulk too largely and require to be supplemented by a little animal food rich in oil, or seeds rich in albuminates or oil, such as ground-nuts, or by adding other pulses less rich in oil and supplying the requisite amount of oil separately.

Some useful notes are given on the decomposition of flesh and fish foods in the Tropics and the danger of such decomposed food giving rise to choleraic diarrhœa, while diseased meat and adulterations of various kinds are also considered.

A good little book is that by MacKnight,[2] which gives lists of foods found in and suitable for the Tropics. It is a careful compilation and useful for reference.

El gofio is a food which has been praised for use in warm climates.[3] It is manufactured from different flours, including those obtained from wheat, maize, barley, white lupine, rye, chick-peas and beans. It is said, along with milk and eggs, to be amply sufficient for nourishment in the Tropics and is recommended as a ration for soldiers.

Wiley[4] mentions the value of a fruit diet and draws attention to the fact that the prevailing characteristics of the natural food of the Tropics is found in the excessive quantity of sugar which they contain, or, as in the case of cassava, of starch. The amount of protein is very small, and the amount of oil is, with the exception of the cocoanut, not marked. On the whole it appears that the natural tropical food is one composed of a large excess of carbohydrates, in which sugar predominates. A gradual change in dietary is indicated when one takes up residence in a tropical climate.

An excellent paper is that by Cantlie.[5] He derides the erroneous belief that the natives of the Tropics and sub-Tropics live on rice, and points out that vegetable eaters, quite apart from the vegetarian faddists, are by no means confined to hot countries. He then considers the various kinds of food available in the Tropics, some of his remarks applying with particular force to the Sudan. He points out why beef is difficult to obtain and why, when obtained, it is often tough and unsavoury. He mentions that most Europeans eat more meat (when they can get it) in the Tropics than at home. Some say this is because the climate is exhausting, and more strengthening food is required; but the real reason, no doubt, is that

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[1] Simpson, W. J. (July 15th, 1903), "Tropical Hygiene." *Journal of Tropical Medicine*, p. 224, Vol. V.

[2] MacKnight, T. M., "Food for the Tropics," London, 1904.

[3] Reyes, S. (March 12th, 1904), "Food in Warm Climates." *Lancet*, p. 739, Vol. I.

[4] Wiley, H. W. (December 3rd, 1904), "Diet for the Tropics." Quoted in Epit. *British Medical Journal*, p. 83.

[5] Cantlie, J. (October 15th, 1906), "Food and Digestion in the Tropics." *Journal of Tropical Medicine*, p. 312, Vol. IX.

Food— *continued*

the nutritive quality of the beef and mutton is inferior, and that more has to be taken to supply the bodily wants, thereby taxing the digestive organs, which in hot climates are usually feeble, and bringing a train of gastric, hepatic and intestinal troubles. As regards fowls, it is stated that their nutritive value is relatively small and chicken is frequently difficult to digest. Moreover, the monotony of eating fowl day after day, and month after month, let the fowl be cooked in ever so many ways, palls upon the appetite after a time, and digestion and nutrition suffer. One may add here that this is still more true of pigeons, which form such a staple dish in Egypt and the Sudan. "Curry," says Cantlie, "if properly made, is a hygienic dish and the condiments should be added by the consumer himself, or herself, at the table and not by the cook." The word "curry" really means sauce, and the therapeutic use of pepper seems to be not so much as a stomachic tonic as an intestinal stimulant and chiefly as a stimulant to the large intestine. It is the large intestine that first flags in its duty in the case of natives of tropical countries, who are very subject to constipation owing to the atonic condition of the colon, a state of things often remedied by the use of black pepper.

Alcohol. The natives of warm climates, both by their religion and their habits, shun alcohol. It is in no sense a food, and Europeans in tropical countries would do well to avoid its use altogether.

Spirits and beer in hot, moist climates are positively detrimental to health; light wines, white or red, do least harm. Champagne, taken after excessive fatigue, about sunset, is perhaps the safest form of alcoholic beverage. It should not be taken with meals, but only on reaching home after a fatiguing march, or long exposure to wet.

It is possible that in such a dry country as the Northern Sudan alcohol is not so detrimental as in moist regions if taken only after sunset, and many feel the better for an evening "nip." At the same time, the evil effects of alcohol may be insidious and may not show themselves for years. On the whole, I am inclined to think that the total abstainer is the more energetic and more healthy man here as elsewhere in tropical countries, but it is well not to be too dogmatic and to remember "what is one man's food is another's poison."

Tea. "As a stomachic tonic," says Cantlie, "and as a safe way of introducing fluid to the system, tea would seem beneficent and hygienic." It was evidently introduced by the Chinese, owing to the calamities arising from drinking unboiled water. Deep well-water is almost unknown in China, and the shallow wells and streams are so apt to become polluted, owing to the habits of the Chinese, that experience dictated the necessity of boiling the water. But boiled water being insipid, and the object of its being boiled not being evident to ignorant and thoughtless people, the water was "flavoured" by the leaves of the tea plant, a custom which has become widespread. It was, no doubt, for hygienic purposes tea was introduced, but the abuse of tea-drinking has brought many evils in its train. The Chinese drink tea after finishing their principal meal, and in fact as a drink at any time. They do not drink tea during their meal, but after the meal is finished. The pernicious system of drinking tea during a meal is one peculiar to British folk, and the habit is fraught with many dyspeptic troubles. The best China tea, prepared by pouring boiling water over the leaves and immediately pouring the water off the leaves, is a wholesome fluid, calculated to aid digestion, especially when taken after the meal is finished. Tea taken with animal food, be it eggs, fish, flesh or fowl, is a certain means of producing dyspepsia, for when the tea is "drawn" for a long time, and when the tea used is of an inferior quality—the method and material usual in Britain and Australia—the tannic acid of the decoction, uniting with the albumen of the animal tissues, produces a leathery compound which no gastric juice, however potent, can penetrate and digest. Tea, used as the Chinese use it, is a hygienic drink; as it is usually used in Britain and by the British folk throughout the Empire it is detrimental to the public health.

Coffee. Two or three mouthfuls of good coffee after a meal are an aid to digestion; taken in quantity—breakfast-cupfuls—it is an impediment to digestion, and, diluted with half milk and taken with a meal of eggs, fish, fowl, or flesh, is still more so.

Tobacco. In moderation, and smoked soon after a meal, the deleterious effects of tobacco are infinitesimal. When indulged in to excess, say six to eight cigars, or fifteen to twenty cigarettes, or 1 oz. of pipe tobacco, it is an injurious cardiac depressant.

An interesting letter is that by Payn,[1] whose conclusions we quote, although he refers more to conditions suitable to cold than hot countries. Still, even in hot countries sugar is

[1] Payn, F. W. (September 21st, 1907), "Athletics and Food Values." *Lancet*, p. 859, Vol. II.

of decided value as a producer of energy, and I have myself felt benefited from taking a liberal allowance of sugar during the trying summer months. I am inclined to think that the good effects of the cup of strong coffee so frequently taken in the forenoon during office hours are due in some measure to the contained sugar. Payn mentions :— **Food—** *continued*

1. The incalculable restorative effects of liquid at a high temperature after over six hours of continuous marching. So great is the effect of boiling water on the efficiency of a man undergoing a forced march of 10 to 14 hours that I feel certain, from my experiments, that it is more important to provide the soldier on a long march with a small apparatus for heating liquid than with food. The extent to which boiling water can take the place of food was never fully realised by me until I marched 14 hours on three sandwiches and plenty of hot water. Nothing but the possession of a spirit-lamp saved me from serious illness from fatigue and exposure during some of these marches, and I have no doubt many travellers could corroborate this.

2. The imperious craving for sugar in some form which these long marches produce and the enormous importance of an adequate supply of sugar in the diet of soldiers performing much bodily exertion. I have no hesitation in saying, firstly, that the importance of sugar (owing to the consumption of the sugar in the blood by bodily exercise) is most inadequately recognised in English military diet; and, secondly, that the private soldier is too often driven to satisfy the natural craving for sugar after violent exercise by drinking alcohol. Hence he believes that alcohol is natural and does him good. I further believe that it could be shown by experiment that men who were allowed a glass of milk with four lumps of sugar in it could undergo greater fatigue on that drink than on almost any other.

3. The vast superiority of hot oatmeal porridge at breakfast and supper over almost every other article of food in maintaining efficiency and health during prolonged marches. Abnormal exertions, such as 15 hours of climbing, throw the real value of foods into a far stronger relief than usual. The presence or the lack of that food, during an ordeal of this sort, at breakfast or supper has so vast an effect on one's condition that I can scarcely imagine any General who is aware of its value overlooking it as an almost complete and most portable food for a forced march of 14 hours. It is far richer in mineral salts than meat.

4. The utility of dried figs. Given a meal of hot porridge for breakfast and supper a soldier could march without discomfort or harm for a whole day or night on a handful of figs and some hot liquid, owing to the fact that they are so full of sugar and mineral salts, which are what the marching man chiefly needs. Anyone who overlooks the value of the fig in catering for the food of an army corps on campaign commits a great blunder. In a rapid campaign the great requisite in food is the irreducible minimum for health and strength, which is not the case in time of peace. I believe that a scientific medical investigation of the effects of the ordinary soldiers' diet and of such a diet as the one indicated above in the case of men undergoing long tests of endurance, would be exceedingly valuable from a military point of view as well as of great scientific interest, and I also believe that a medical investigation of the dietetic tastes of persons who are known to perform so much bodily exertion as the leading lawn-tennis players could scarcely fail to disclose new and valuable facts on the relation of athletics to food values.

As regards tinned foods, Cathcart[1] deals with the bacterial flora found in "blown" tins, chiefly in those containing sardines. The tins were bulged and, on being opened, a fœtid gas escaped, but the flesh of the sardines appeared quite normal and healthy. It was found that organisms of an intestinal type were present, which on re-inoculation into sound tins gave rise to a gaseous decomposition. No toxic symptoms were produced on feeding guinea pigs with the contents of the "blown" tins.

Beans form a favourite article of consumption in the Sudan, hence attention may be directed to an epidemic of poisoning due to their use when tinned, and recorded by Rolly.[2*] *Bacillus paratyphi*, B., and *Bacterium coli commune* were found present, but, owing to the fact that the beans had been heated almost to the boiling-point, the illness was of a very benign character. Two hundred and fifty people, were, however, affected. The bacteria appear to have been killed and only their toxins consumed.

A question sometimes asked in the Tropics is—"How long may tinned foods be expected to remain in good condition?" Harrington[3] answers this by stating that properly canned foods, according to the evidence at hand, should remain in good condition indefinitely. He cites a case where tins were known to remain in good condition for 63 years. At the same time, there do not seem to be any statistics on this point so far as hot countries are concerned.

Beveridge,[4] has an instructive paper on South African experiences. He found that no tinned meat stocked in the open, exposed to changes of temperature, heat of the sun and effects of rain in warm climates, should ever be kept for more than one year. When under suitable cover, perhaps for two years, but never more, and in all cases should be inspected at intervals. He explains that the paint of the tins gets cracked or knocked off, damp and heat induce rust which specially affects dirt or cracks, and a hole, which may be very minute, speedily forms. He also notes that, on long keeping, a change, of the nature of adipocere, not understood, sometimes takes place in the meat itself, and this is another argument against long keeping. Paper labels are condemned, while only painted tins should be accepted.

[1] Cathcart, E. P. (August, 1906), "The Bacterial Flora of 'Blown' Tins of Preserved Food." *Journal of Hygiene*, p. 248.

[2] Rolly, *Münch Med. Woch.*, 1906, No. 37, p. 1798.

[3] Harrington, C. H., "Practical Hygiene." 3rd Edition.

[4] Beveridge, W. W. O. (August, 1906). *Journal of the Royal Army Medical Corps.*

* Article not consulted in the original.

Food— *continued*

The most dangerous tinned foods are those containing much moisture, *i.e.* milk, salmon, lobster and mixtures of meat and vegetables.

The more acid foods, such as fruit, jams and vegetables, are more liable to take up metals from the tins. The simpler the preparation, the better it stands the effects of climate and heat. Useful notes on inspection are given. Apparent bulging may be due to the tins being dented. A good tin of meat has usually slightly concave ends owing to a partial vacuum forming during the process of sterilisation.

Re-soldering should be looked for. As a rule, two holes are made in one end of the tin to permit steam to escape. Re-soldering, or the presence of a third or more soldered holes points to puncture to allow gas to escape.

Dented tins, if otherwise fit, should be issued early, as they are apt to rust and perforate on keeping.

On opening certain tins, *i.e.* of marmalade, rhubarb, tomato soup, etc., a blackened appearance may be noticed. This is due to the action of the vegetable acids on the tin-plating, and if slight, and there is no evidence of fermentation as evidenced by minute gas bubbles, may be neglected.

Decomposition may result from incomplete sterilisation, or incomplete sealing of the tin. Bulged tins, may be tested by puncturing them under water to test for the escape of gas. In some cases, a little gas will escape from tins containing perfectly sound meat, owing to incomplete exhaustion during the process of sterilisation, but which, being sterile, is of no real consequence and amounts to, as a rule, only about 1 c.c. or so. One test described is as follows:—When the swelling is not apparent, the tins are boiled for one hour, which causes, by expansion, the ends of all to swell; they are then cooled and set aside for eight hours, when the sound ones will return to their former condition. The unsound ones will remain bulged as the convexity is due to the pressure of gases. Viry states that putrefaction may take place in tinned meats without the formation of gas, but Beveridge has not been able to confirm this. The presence of moulds at once condemns, the sterilisation not having been efficient. Moulds impart an unpleasant taste to the food and are apt to cause diarrhœa.

Eber's test for the decomposition of meat is said to be useful but not absolutely reliable, owing to the presence of trimethylamine, in, for instance, mutton and pickled foods. A small quantity of the reagent, which consists of one part sulphuric ether, one part pure HCl. and three parts ethylic alcohol, is placed in a test-tube or other suitable vessel. The material to be examined is smeared on the end of a glass rod, which is dipped below the surface of the reagent but is not allowed to touch the side. If ammonia be present, a cloudiness appears or fumes may be given off.

Food Poisoning. This is a subject of very considerable importance in all hot countries, and one has seen several examples of it in the Sudan. It may result from:—

1. Faulty preparation of food, as from dirty kitchen utensils, the dirty hands of cooks and their assistants, imperfect or defective cooking, or the addition of deleterious substances, either designedly or accidentally.

2. Decomposition which is very apt to occur, especially in foods kept over-night.

3. Contamination, apart from preparation, *i.e.* from faulty storage or from the filthy feet of flies or other insects.

4. Injurious food-stuffs, such as bad tinned foods or imperfectly cured or preserved foods.

I recall an epidemic occurring at the Grand Hotel, Khartoum, and in this connection Walker's paper[1] on the so-called "Canary Fever" of Las Palmas is specially interesting. He has shown that this condition is in all probability due to bacterial infection of food. It is peculiar by occurring in hotels, coming on suddenly, and attacking a number of hotel residents at the same time. It is characterised by vomiting or nausea, followed by diarrhœa, and the stools may contain mucus and even blood. The temperature may rise, but not as a rule to any considerable extent. The length of attack varies from two days to three weeks. The causes are discussed, and I quote here in full the preventive measures recommended, because I think they are specially applicable to the hotels and numerous restaurants in Khartoum, and because it has been found necessary in certain cases to enforce the adoption of somewhat similar precautions:—

Meat and fish, particularly, should be protected from flies in as effective a manner as possible before it is brought into the hotels.

When in the hotels all food should be protected from flies; the larder should be entirely fly-proof; the entrance should be protected by two doors, between which there is room for a man to stand; both these doors should close automatically with springs, and it would be well to have some simple automatic arrangement which would prevent one being opened until the other was closed. It should be easy to catch the few flies that might possibly get into the larder, in spite of these precautions, by means of fly traps.

Of course the best plan would be to keep the food in a chamber which was constantly below freezing-point. When the food was removed, once or perhaps twice during the day, it should be kept in fly-proof receptacles.

Meat should be kept hanging up, and not laid upon shelves.

Shelves and tables in the larder, serving rooms and kitchens, should be made of some non-absorptive material, such as marble or slate. Most of the shelves and tables upon which the food was placed during the process of cooking and serving, which I saw in the islands, were made of soft wood. No matter how much this wood be

[1] Walker, C. E. (February 29th, 1908), "Observations on the so-called 'Canary Fever.'" *Annals of Tropical Medicine and Parasitology*, p. 483, Series T.M., Vol. I., No. 4, Liverpool.

Food Poisoning— *continued*

scrubbed, there must always be a certain amount of organic material in a more or less advanced stage of decomposition in the cracks. In the serving rooms, kitchens, etc., and wherever food is exposed for any length of time to contagion by flies, the food should be covered up as soon as it is put down. The ordinary wire gauze dish covers are cheap, and admirably suited to this purpose.

Cooking utensils, plates, dishes, forks, spoons, etc., should be sterilised shortly before use. This would not involve any very considerable extra labour, and convenient apparatus would not be very costly. No pressure of steam would be necessary, only the utensils should be brought to the temperature of steam. Cleaning with a jet of live steam, such as is done on ships, would be very effective.

Copper cooking utensils have the disadvantage that they require re-tinning at intervals. There is no means of getting this done in a first-class manner in the islands. The tinning is often irregular, and it is impracticable to get such a surface really clean and free from small collections of organic material. Something other than copper would, therefore, be an advantage.

Soup must be made fresh every day, and the stock-pot abolished.

With regard to rechauffés, even if protection from flies is guaranteed between the first and second cooking, it would be well if the material were always brought to boiling-point and kept so for some minutes.

Cold cooked provisions must be kept free from flies.

There should be but little difficulty in keeping the kitchen, and even the whole house, comparatively free from flies by means of wire gauze frames to the windows and double doors; the outside door to consist of a frame with wire gauze stretched upon it. Such a plan would allow plenty of air to come into the rooms, and would exclude the majority of the flies. This is done very extensively in America, and even by some people in England.

No suggestion is intended that the kitchens of the hotels are not clean in the ordinary acceptance of the word. For instance, the kitchens of those I visited would compare very favourably with any kitchen I have seen in Europe.

What the observations really imply is that precautions which are sufficient in England to prevent a degree of infection by bacteria enough to produce symptoms, are wholly inadequate under the conditions of temperature, etc., in the lower and hotter parts of the islands.

It is quite possible that there may be one or more specific bacteria which are specially responsible for the acuteness of the symptoms. Even if this be the case, however, there seems but little doubt that the flies are to a large extent responsible for the original infection of the food. The rapid multiplication of the bacteria and the consequent production of toxins depends upon the local conditions. It would seem that the suggested precautions are necessary whether there be a specific micro-organism or not. It is probable that food is more frequently infected, even in the best conducted private kitchens in the towns in the islands, than is the case in Europe, and that consequently the residents may have acquired a limited degree of immunity. I met several residents, however, who told me that they had suffered from attacks after dining at hotels, but not at any other time.

An important paper, dealing with the bacteriological aspects of an epidemic of food poisoning due to brawn containing the *Bacillus enteritidis* of Gærtner, is that by Buchan.[1] It is likely to be useful to any bacteriologist having to carry out an investigation of an outbreak of this type.

Titze[2] sums up our present knowledge regarding meat poisoning as follows:—

1. By far the majority of cases of meat poisoning hitherto investigated have been shown to be due to bacteria belonging to Gærtner's group or to the paratyphoid *B.* group.

2. These bacteria usually obtain entrance to the tissues of animals intended for slaughter as a result of septic disease. They may not be the primary cause of septic processes, but possibly constitute an accompaniment of the general disease condition produced by ordinary sepsis-producing organisms.

3. The paratyphoid bacillus may also be conveyed to the flesh of perfectly healthy animals through various accidental circumstances (poisoning by sausage meat).

4. We know nothing regarding the occurrence and spread of meat poisoning bacilli in and by healthy men and animals, or their mode of existence outside the animal body; we are equally ignorant regarding the reasons for the variation in their powers of producing toxin, and in regard to the essential factors in toxin production.

5. No sufficient investigations have been conducted regarding the injurious qualities of meat which has undergone albuminous decomposition in consequence of the action of saprophytes (ptomaines and sepsins).

6. Botulismus is produced by an anærobic saprophyte, the *Bacillus botulinus*.

Guinea Worm (Dracontiasis). The most important recent work on this subject is that by Leiper.[3] He first of all classifies the hypotheses of infection that have been advocated, as follows:—

i. Those in which the development of the embryo is supposed to occur without the intervention of any intermediate host, human infection being caused by—

(a) The embryo, as discharged from the parent worm; or

(b) The mature larva, evolved from the embryo in water or marshy soil; or

(c) The young adult, the product of the continued growth of the larva in water.

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[1] Buchan, F. (December 7th, 1907), "An Outbreak of Food Poisoning due to Eating Brawn." *Lancet*, p. 1604, Vol. II.

[2] Titze, C. (March, 1908), "Zeits für Fleisch und Milchhyg." Quoted in *Journal of Comparative Pathology and Therapeutics*, March, 1908, p. 87.

[3] Leiper, R. T. (January 19th, 1907), "Etiology and Prophylaxis of Dracontiasis." *British Medical Journal*, p. 129, Vol. I.

Guinea Worm— *continued*

He points out that these theories have become discredited, and gives further experimental and literary evidence in favour of their being discarded. The embryos cannot infect man by the skin or mouth or undergo further development in water. His own observations on the vitality of the embryos in water show that the usual period of survival was three days, though some survived till the sixth day. In mud they lived a day or two longer, probably because in this medium they move more slowly and are consequently exhausted. Although provided with a mouth and digestive tract they are still unable to obtain food for themselves.

ii. Those in which an intermediate host is considered essential for the development of the larva in order that it may become fitted to re-infect man.

(d) The only, and in itself sufficient, host being cyclops.

(e) A second, and at present unknown, intermediate host being necessary to continue and complete the changes begun in cyclops.

Leiper, in his experiments in Nigeria, found that, of all the organisms in the ponds, cyclops alone was capable of infection, and he believes that the mode of entry of the embryo is not through the integument of the cyclops, as usually taught, but by way of the intestine. As regards the completion of metamorphosis, he found that the striated cuticle of the embryo was cast generally on the eighth day. The larva which emerged lost, two days later, a very delicate enveloping pellicle, and from that time onwards underwent no further ecdysis. The subsequent changes were confined to the differentiation of internal structures, the larva apparently becoming mature in the fifth week. These observations differ from those hitherto accepted.

Leiper also noted that the larvæ showed no disposition to leave the cyclops and become free-swimming, evidence that infection of man does not occur by the skin. As time went on the larvæ became more quiescent, and when the cyclops died the larvæ were found dead in its interior.

As regards the way in which they leave their host, Leiper[1] refers to his previous work, in which he demonstrated the action of a 0·2 per cent. solution of hydrochloric acid in killing the cyclops and rousing the larvæ to such activity that they speedily escaped by the mouth, anus, genital opening, or a breach in the cuticle of the cyclops, and swam about freely in the fluid.

The later work has consisted in feeding a monkey on bananas containing cyclops which had been infected for five weeks, and which had in them apparently mature larvæ. Six months later a careful post-mortem examination of the monkey revealed the presence in the connective tissues of five filariæ, which possessed the anatomical characteristics of *Filaria medinensis*. There were three unimpregnated and obviously immature females about 30 mm. long, and two remarkably small males (22 mm.), which were obtained one from the psoas muscle and the other from the connective tissue behind the œsophagus. These results (says Leiper) point strongly to the truth of the theory that infection of man takes place from the drinking of water containing infected cyclops. The suggestion that a second intermediary host is necessary for the complete development of the guinea worm larva is disposed of by the fact that this is actually attained in cyclops.

He comments upon the importance of the discovery of the male and immature female forms in the connective tissue, showing that the guinea worm thus comes into line with what we know of the after-development of other filariæ.

He thinks that Geotropism (tendency to grow downwards towards the earth) affords a rational explanation of the remarkable distribution of the parasite in man.

An important fact to which attention is drawn is that the embryos are immediately killed if dried by natural evaporation, and they cannot be revived by the re-addition of water.

A review is given of the conditions essential for the completion of the life-cycle of the parasite, as follows:—

The young must be discharged directly into fresh water soon after the parent worm has succeeded in creating a break in the overlying skin and before the wound has become markedly septic. The embryos must find a cyclops within a few days. They must, moreover, succeed in entering its body cavity. Five weeks later they will have developed into mature larvæ. These must, thereafter, be taken into the human stomach, and having been set free

[1] Leiper, R. T. (January 6th, 1906), "The Influence of Acid on Guinea Worm Larvæ encysted in Cyclops." *British Medical Journal*, p. 19, Vol. I.

from their host by the gastric juice, reach the connective tissues by penetrating the gut wall. The life-cycle of the parasite will necessarily be broken:— **Guinea Worm—** *continued*

(1) By the death of the embryos, either from sepsis while still within the parent worm, or, if after their discharge, by saltish water or drying. (2) If cyclops are not present in the water or, if the infected cyclops die or are not taken into the human stomach. (3) If the larvæ, ingested by the final host, are immature or fail to escape from the chitinous sheath of the cyclops. Though they do find their final habitat, the cycle will still be incomplete if (4) there are not both males and females among the matured adults and if in their wandering the females are not impregnated.

It will at once be seen from the above summary that the isolation of infective man from healthy cyclops and of infected cyclops from man must be the object of any organised effort to stamp out dracontiasis.

Leiper then proceeds to detail preventive methods, so far as the West Coast of Africa is concerned. These really resolve themselves into prevention of the fouling of water and the provision of pure water. They have more or less a local bearing, but he mentions that he found another nematode larva in cyclops which might be a source of error in the course of investigations. Finally, in a suggestive paragraph, he indicates lines of future work. We tabulate the various points:—

1. Accurate observations as regards the conditions under which the intermediate host lives and multiplies in tropical countries.

2. Determination of its natural enemies.

3. Determination of its food supply.

4. Observations as to whether it can survive the drought of the summer, buried beneath the sun-caked mud, or if, when once a pool has dried, it must be re-stocked from another source.

5. Experiments to see if, by the addition of chemicals, we can destroy the cyclops in suspected waters without rendering these useless or dangerous to man.

As if in answer to these suggestions by Leiper, we find papers by Graham and Brady[1] on the Cyclopidæ of the African Gold Coast. The former points out that the inference that all species of Copepoda cannot act as efficient hosts to guinea worm is strengthened by the following considerations:—

1. There is a large number of species.

2. The habits of the different species vary greatly. Some are surface feeders, some are found at the bottom. Some inhabit foul, some clean water. Some leave the water to climb on stalks of water-weed enveloped in a drop of water carried with them, some do not leave the water; some are found in streams, some are not.

3. The different species are infested by different parasites, some only by ecto, others by ento-parasites (worms).

4. The different species differ in the date of their appearance in the pools. Some are found early in May; some appear, or, at least, only become numerous, in July.

The significance of the date of appearance is dealt with in a previous paper,[2] where it is shown that, in the Gold Coast, June is the month during which the signs and symptoms of guinea worm infection attain their maximum. Presumably the month of maximum manifestation in man is the month of maximum infection of cyclops. It is pointed out that in the locality examined, when the streams were full of water, cyclops were found in the streams and not in the wells, but when the channels were dry the cyclops occurred in the wells and bred there. The natives stand in the wells during the dry season whilst drawing water; the embryos in their legs can then escape and infect the cyclops. The prophylactic measures recommended are (1) careful filtration of the drinking water; a fine handkerchief will serve the purpose, as by this means cyclops can be readily removed; (2) prevention of infection of cyclops by providing troughs for the natives to draw water from, so that the cyclops is excluded.

In a discussion on the above remarks, Sandwith stated that in the Sudan human beings suffered from *Filaria medinensis* during two or three months of the year only, there being a distinct periodicity. Chalmers stated his belief that the great amount of physical infirmity due to guinea worm was not fully appreciated.

The life-span of the female worm, as noted by Manson and others, extends to about one year (Graham says roughly ten months), and evidently depends on the habits of the species of cyclops which serves as its intermediate host.

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[1] Brady, G. S. (November 9th, 1907), "Notes on Dr. Graham's Collection of Cyclopidæ from the African Gold Coast." *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. I., No. 3, p. 423.

[2] Graham, W. M. (August 15th, 1905), "Guinea Worm and its Hosts." *Journal of Tropical Medicine* p. 248, Vol. V.

Guinea Worm— *continued*

So far as the Sudan is concerned, Bray[1] has shown that it is doubtful if dracontiasis exists in Kassala, but it occurs in Gedaref and is very common in Gallabat on the Abyssinian frontier. It occurs but with no great frequency on the blue Nile, but is common on the Upper White Nile, and is very prevalent in the Bahr-El-Ghazal Province and in Kordofan. It is found chiefly in villages using surface water or shallow wells or employing hollow Tebeldi trees *(Adansonia digitata)* as water reservoirs.

He notes that the life-span of the female worm is from nine months to one year. Captain Cummins recommended that each native soldier be provided with a strainer like that mentioned by Graham. That the Nubas of Kordofan believe that infection takes place by way of the skin is shown by the fact that they wear wooden pattens when crossing wet or marshy places, whence, as experience has taught them, infection may be derived. (*See* Captain Anderson's paper, Third Report.)

Hæmatozoa. This is a big subject and the literature upon it is very scattered, but a good *résumé* by Sambon will be found in the 4th Edition of Manson's Tropical Diseases. His new classification is also given, but whether it will stand the tests imposed by time and increased knowledge remains to be seen. The statement that the oökinete of the Hæmogregarinidæ encysts and produces sporozoites in secondary cysts or spore bags was, I believe, founded on Christophers' work[2] with *Hæmogregarina gerbilli* and lice. The latter, however, has had reason to doubt the correctness of his observations regarding the stage in the louse, and believes that the appearances he described were due to a coccidial infection of the louse itself.

Dutton, Todd and Tobey[3] describe certain parasitic protozoa observed by them in the Gambia and Congo Free State, amongst which we note the following blood parasites:—*Trypanosma theileri*, in all probability occurring in antelopes as far north as Kasongo, several other forms of trypanosoma, including a large one in the monkey *(Cercopithecus schmidti)*, malarial parasites in monkeys, malarial parasites and possibly spirochætes in bats. They also found free gregarine forms in the blood of healthy dogs, probably the vermicule stage of a hæmogregarine similar to that found in dogs in India, and also in dogs in Khartoum, by Captain Olver, P.V.O., but so far undescribed.

In birds Proteosoma was never seen, but Halteridium was very common. This is interesting in the light of Dr. Wenyon's investigations in the Southern Sudan.

So-called Leucocytozoa of birds are described, and this subject will be found considered by Dr. Wenyon, whose observations are at variance, as regards the morphology of these parasites, with those of the Liverpool observers.

Various parasites in reptiles and amphibians are described and figured. What is called a Cytamœba of the frog appears to me to resemble very closely the endoglobular forms of spirochæte I have found in fowls and geese in Khartoum. One cannot enter into any details regarding these numerous hæmatozoa, but some of the more important, such as trypanosomes and spirochætes will be considered under their appropriate headings.

The most interesting of recent discoveries is that of the Sergents in Africa, who in a patient suffering from night-sweats and nausea found on two different occasions a peculiar ectoglobular parasite. It is vermiform in shape and measures about 40 μ in length by 1 to 1·5 μ in size and is sharply pointed at either end. What seems to be a nucleus occupies the whole of the middle third of the body. It was noticed that the parasite used to disappear from the blood about 6 p.m. It therefore possessed a periodicity like filaria embryos. A sketch of this parasite is given in the latest (3rd Edition) of Christophers' and Stephens' work.[4] In this volume will be found notes on practically all the hæmatozoa found up to date, and it is perhaps scarcely necessary to mention more of them, with the exception of the hæmogregarine of cattle, *H. bovis*, found in Erythrea by Martoglio and Carpano.[5*]

[1] Bray, W. (October, 1904), "The Southern Sudan: Its Climate and Diseases." *Journal of the Royal Army Medical Corps.*

[2] Christophers, S. R., "Hæmogregarina Gerbilli." *Scientific Memoirs of the Government of India*, No. 18. Calcutta, 1905.

[3] Dutton, J. E., Todd, J. L., and Tobey, E. M. (November 9th, 1907), "Concerning certain Parasitic Protozoa observed in Africa." Mem. XXI, Liverpool School of Tropical Medicine, and *Annals of Tropical Medicine and Parasitology*, Vol. I, No. 3, p. 285.

[4] Stephens, J. W. W., and Christophers, S. R., "Practical Study of Malaria," p. 261. 3rd Edition.

[5] Martoglio, F., and Carpano, M., *Ann d'Ig. Sperim.*, t. XVI., 1906.

* Article not consulted in the original.

This was a single observation, and it seems a little doubtful if the interpretation was correct. The parasites were 7 μ to 10 μ in length and from 1 μ 6 to 2 μ in breadth, and had rounded ends. Inoculation experiments failed. Mention may also be made of the *Spirochæta theileri* found in cattle in the Transvaal and the Cameroons, the *Sp. ovis* of sheep in Erythrea, which is possibly identical with the *Sp. theileri* and the *Sp. equi* which occurs in mammals in the French Sudan.

Sambon[1] has drawn attention to certain appearances in the hæmogregarines of snakes, namely, delicate oblique lines passing transversely across the long axis of the parasite at from 1 μ to 2 μ from one or both of its extremities. He regards these as possibly representing lines of future cleavage of the capsule of the sporont, sporont being the term applied to the new forms developed from the merozoites and destined to pass into the body of the definitive host and so carry on the further life of the parasite. He also describes a beak-like projection at the anterior extremity of the sporont, and in one species noted a definite dimorphism which may represent sexual differentiation.

Two other discoveries may be quoted, as their confirmation might well be worked out in the Sudan. These are (*1*) the observations by the Sergents[2]* that *Hæmoproteus* (Halteridium) *columbæ* passes through its stage of sporogony in one of the *Hippoboscidæ*, *Lynchia maura*. The incubation period in the pigeon is from 34 to 38 days, and the earliest forms in the bird's blood are very minute, *i.e.* 1 μ to 2 μ in diameter. (*2*) The confirmation of this observation as regards *Hippoboscidæ* by Aragao,[3]* and his statement that part of the cycle of evolution is passed in the lung of the pigeon, cysts containing the merozoites being found in the large mononuclear leucocytes of the pulmonary capillaries.

Heat Stroke. Duncan[4] describes the clinical varieties as follows:—

A. Heat collapse. B. Heat stroke. (*a*) Direct heat stroke or sunstroke proper; (*b*) Indirect heat stroke.

A. Heat collapse. The patient suddenly turns giddy and falls. Skin moist and cool. Breathing hurried but never stertorous, pulse small and soft, pupils dilated, temperature at or below the normal. No loss of consciousness, and recovery the rule.

B. Heat stroke (*a*). Direct heat stroke or sun stroke. There are several forms. 1. Occurs in persons unaccustomed to marching and attacks them specially when the air is moist. There is violent headache and oppression followed by convulsions, loss of consciousness, difficult respiration, small and irregular pulse and often incontinence of urine.

2. Is characterised by excessive sweating, pallor, cyanosis, shallow breathing, injected eyes, swollen veins and partial collapse without complete unconsciousness. Revival occurs under proper treatment.

3. No fatigue is complained of, but the patient is thirsty and suddenly falls forward comatose. The coma may last 24 to 36 hours and end in death.

4. After exertion and exposure to the sun a racking headache sets in. This becomes intense and finally agonising. Great intolerance of light ensues, followed perhaps in 48 hours by unconsciousness. If death does not occur, the intense pain in the head may last from six to eight weeks unrelieved by any drug, but there may be slight evening remissions. It then gradually abates. (*b*) Indirect heat stroke. This is the syncopal form, occurring not in the open but in the hot house or bungalow. Duncan finds it the most frequent type. At the onset the skin becomes pale; there is nausea, colic and incontinence of urine. Convulsions now follow, to be succeeded by cyanosis, dyspnœa and insensibility. The breathing is stertorous, the pupils contracted and the body temperature may reach 108° F. to 110° F., remaining high post mortem.

I have seen such a case, terminating fatally, in a young British soldier in Khartoum. The diagnosis at first was very difficult, renal colic being the condition which suggested itself. The post mortem appearances, especially a peculiar bluish and milky opacity of the brain membranes, recalled another case which was not diagnosed during life and which was complicated by a form of irritant poisoning. I have known type No. 3 occur in Khartoum, but I am inclined to think, from what I can gather, that heat stroke is rare in the Sudan, doubtless in part because of the excessive dryness of the atmosphere throughout the greater part of the summer. Dr. Crispin notes that it is commoner on the moist Red Sea Coast.

Duncan deals with the indirect causes and considers treatment under Preventive and Curative Measures. As regards the former, he mentions the custom, common to old European residents in Egypt, of wearing under the helmet, a tight jean skull cap similar to that worn by the Arabs under the turban or tarboosh. I have never heard of this custom

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[1] Sambon, L. W. (June 15th, 1907), "Hæmogregarines of Snakes." *Lancet*, p. 1650, Vol. I.

[2] Sergent, Ed. & Et. (November 24th, 1906). *C. R. Soc. Biol.*, t. LXI.

[3] Aragao, de B., *Brazil Medico*, t. XXI., No. 31, August 15th, 1907. Quoted in *Bull. de l'Instit. Pasteur*, November 15th, 1907.

[4] Duncan, A. (April 1st, 1903), "On Heat Stroke." *Journal of Tropical Medicine*, p. 101, Vol. V.

* Article not consulted in the original.

Heat Stroke *—continued*

being in vogue in the Sudan. Proper forms of helmet, tinted glasses, loose clothing of a proper colour, and the spinal pad are all considered.

As regards treatment, douching the head and neck with cold water, the application of ammonia to the nostrils, turpentine enemata and mustard poultices to the chest are mentioned. The use of ice to the head is contra-indicated where the skin is cold and the pulse feeble. Convulsions indicate a few whiffs of chloroform. In cases of direct heat stroke in Italy, trinitrin has been found useful, a solution of 1 in 1000 being given in doses of 20 minims to 4500 minims of water every quarter of an hour. Venesection is dangerous.

Manson quotes Chandler's treatment for hyperpyrexial cases. It consists principally in the use of ice and iced water externally, with the patient on a stretcher, digitalis being given to ward off heart-failure. Strychnine is contra-indicated. Artificial respiration has saved cases in desperate straits.

Gardini,[1] describing cases in Florence in 1905, notes that the attacks frequently came on after a full meal when the production of CO_2 was increased, and that the coma of sunstroke resembles that of uræmia, but, unlike the latter, is usually associated with hyperpyrexia. The types he gives in order of frequency are: 1. Mixed forms, 2. Asphyxial, 3. Syncopal, 4. Cerebro-spinal. In every case, he states, the prognosis should be reserved, as cases beginning very slightly may rapidly get worse.

Rogers believes that under the terms Heat Exhaustion, Sunstroke and Heat Stroke or Siriasis, two broadly different conditions are included. First,—syncopal attacks due to exposure to the direct rays of the sun or to hard labour during great heat (*i.e.* in stokeholds of Red Sea and Persian Gulf steamers). In these cases there may be no marked elevation of body temperature, and, if properly treated, recovery is the rule, with or without some permanent mental injury.

Second,—true heat stroke with hyperpyrexia and acute pulmonary congestion, coming on very suddenly, usually without any actual exposure to the sun's rays. Such cases only occur under very trying atmospheric conditions, either excessive dry heat or lesser degrees of moist heat. This is true heat stroke.

In the first class, it is faintness due to heart-failure under special stress which takes place. In the second, it is essentially loss of consciousness due to hyperpyrexia, the cause of which is attributed either to exposure, to excessive heat, producing in some way not yet fully understood, failure of the heat-regulating mechanism of the body, or to the toxins produced by a hypothetical microbe. It may be said at once that Rogers has no faith whatever in Sambon's microbic theory, and adduces arguments against it. He dwells upon the important part the presence of atmospheric moisture plays by checking surface evaporation. Alcohol seems both to predispose markedly to heat stroke and to greatly increase the gravity of the cases. Rogers also contradicts the statement that the disease is never found at an altitude above 600 feet, and shows that 71 per cent. of 424 Indian cases occurred at over this elevation above sea level.

As regards premonitory symptoms, *the desire to micturate freely* receives special mention. It appears to be a valuable warning sign. The author thinks that quinine, guarded by cardiac tonics, should always be used, as, apart from the question of malaria, it is likely to help in restoring the control of the heat-regulating mechanism. He suggests careful intravenous administration, and the rubbing of 10 to 15 minims of creosote into the axilla, as a method of producing diaphoresis. The occurrence of mild forms of fever due to heat stroke is considered, and it is stated that they quite possibly exist but are not common, in Calcutta at least.

Hydrophobia. This disease is happily not common in the Sudan, but a case did occur in Khartoum, and, as in most tropical countries, it may assume importance, some of the recent work upon it—mostly foreign—may be cited.

Williams and Lowden[2] carried out original work with two ends in view.

1. To determine the value of the "Negri bodies" in diagnosis and methods for their rapid identification.

2. To determine their precise nature.

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[1] Gardini, O., *Clin. Modern*, No. 22-24, an XII. Quoted in Epit., *British Medical Journal*, October 6th, 1906.

[2] Williams, A. W., and Lowden, M. M. (May 18th, 1906), "The Etiology and Diagnosis of Hydrophobia." *Journal of Infectious Diseases*, p. 452.

They detail the technique both for smears and sections. That for smears may be quoted here as likely to prove useful:—

1. Glass slides and cover-glasses are washed thoroughly with soap and water, then heated in the flame to get rid of oily substances.

2. A small bit of the gray substance of brain chosen for examination is cut out with a small, sharp pair of scissors and placed about one inch from the end of the slide, so as to leave enough room for a label. The cut in the brain should be made at right angles to its surface and a thin slice taken, avoiding the white matter as much as possible.

3. A cover-slip placed over the piece of tissue is pressed upon it until it is spread out in a moderately thin layer, then the cover-slip is moved slowly and evenly over the slide to the end opposite the label. Only slight pressure should be used in making the smear, but slightly more should be exerted on the cover-glass toward the label side of the slide, thus allowing more of the nerve tissue to be carried farther down the smear and producing more well-spread nerve cells. If any thick places are left at the edge of the smear, one or two of them may be spread out toward the side of the slide with the edge of the cover-glass. If the first smear does not seem to be well spread out others should be made until a satisfactory one is obtained.

4. For diagnosis work such a smear should be made from at least three different parts of gray matter of the central nervous system: first, from the cortex in the region of the fissure of Rolando or in the region corresponding to it (in the dog, the convolution around the crucial sulcus); second, from Ammon's horn; third, from the cerebellum. In many of the animals reported here smears were made from the gray matter of the cerebral cortex, around the fissures of Rolando and Sylvius, from the olfactory bulb, Ammon's horn, cerebellum, medulla in the region of the roots of the cranial nerves, spinal cord in the dorsal and lumbar regions, spinal and Gasserian ganglia, salivary glands, suprarenals, and some of the peripheral nerves. From the last four-named structures the smears were not very successful, so only a few were made.

5. The smears were dried in air, and subjected to one of the two following staining methods:—

(*a*) Giemsa's Solution. The smears are fixed in methyl alcohol (commercial is just as good as pure) for about 5 minutes. The staining solution recommended last by Giemsa (1 drop of the stain to every c.c. of distilled water made alkaline by the previous addition of one drop of a one per cent. solution of potassium carbonate to 10 c.c. of the water) is poured over the slide and allowed to stand for one-half to three hours. The longer time brings out the structure better, and in 24 hours well-made smears are not overstained. After the stain is poured off, the smear is washed in running tap-water for one to three minutes, and dried with filter paper. If the smear is thick, the "bodies" may come out a little more clearly by dipping in 50 per cent. methyl alcohol before washing in water, then the washing need not be as thorough. By this method of staining, the cytoplasm of the "bodies" stains blue and the central bodies and chromatoid granules stain a blue-red or azur. Generally the larger "bodies" are a darker blue than the smaller, the smallest of all may be very light. The stain varies somewhat according to the thickness of the smear. Some have a robin's egg blue tint, but this is after a longer fixation in the methyl alcohol. In this case the red blood cells may have a greenish tint. The cytoplasm of the nerve cells stains blue also, but with a successfully made smear the cytoplasm is so spread out that the outline and structure of most of the "bodies" are seen distinctly within it. The nuclei of the nerve cells are stained red with the azur, the nucleoli a dull blue, the red blood cells a pink-yellow, more pink if the decolorisation be used. The "bodies" have an appearance of depth, due to their slightly refractive qualities.

For diagnostic purposes this method of staining may be shortened as follows: Methyl alcohol, five minutes, equal parts of the Giemsa solution and distilled water, 10 minutes. In this way "bodies" are generally brought out well enough for diagnosis, and sometimes the structure shows distinctly. It is always well, however, to make smears enough for the longer method of staining, in case the shorter one should prove unsatisfactory.

(*b*) The eosin-methylene blue method recommended by Mallory. The smears are fixed in Zenker's solution for one-half hour, after being rinsed in tap-water they are placed successively in 95 per cent. alcohol iodine one-quarter hour, 95 per cent. alcohol one-half hour, absolute alcohol one-half hour, eosin solution 20 minutes, rinsed in tap-water, methylene-blue solution 15 minutes, and dried with filter paper. With this method of staining the cytoplasm of the "bodies" is a magenta, light in the small bodies, darker in the larger; the central bodies and chromatoid granules are a very dark blue, the nerve cell cytoplasm, a light blue, the nucleus a darker blue, and the red blood cells a brilliant eosin pink. With more decolorisation in the alcohol the "bodies" are not such a deep magenta and the difference in colour between them and the red blood cells is not so marked.

The "bodies" and the structure are often more clearly defined with this method, and perhaps, on the whole, it is better to use it for making diagnosis; but when there are only tiny "bodies" present, or when the brain tissue is old and soft, the Giemsa stain seems to be the more successful; above all, when one wishes to study the nature of the central structures and granules the Giemsa stain must be used. We therefore recommend strongly the use of both methods. Even if both are used, and one has to wait for the longer method, the technique is far simpler than any so far published.

Van Dieson, working in our laboratory, suggests a staining method which differentiates the "Negri bodies" more quickly than either of the two methods described above. So far, the best proportion of the stains used have not been determined, but satisfactory results have been obtained from the following mixture: To 10 drops of distilled water three drops of a sat. alc. sol. of rose-anilin-violet and six drops of Lœffler's solution of methylene blue are added. The smears are fixed, while moist, in methyl alcohol for one minute. The stain is then poured on, warmed until it steams, poured off, and the smear is rinsed in water and allowed to dry.

The cytoplasm of the "bodies" is a deep and distinctive red, their inner structures are a dark blue, the nerve cells are a light blue, and the blood cells a pale salmon-red.

The staining mixture remains good for about an hour.

Their summary and conclusions are as follows:—

1. The smear method of examining the "Negri bodies" is superior to any other method so far published, for the following reasons: (*a*) It is simpler, shorter and less expensive; (*b*) the "Negri bodies" appear much more distinct and characteristic. For this reason, and the preceding one, its value in diagnostic work is great; (*c*) the minute structure of the "Negri bodies" can be demonstrated more clearly; (*d*) characteristic staining reactions are brought out.

Hydrophobia *—continued*

2. The "Negri bodies," as shown by the smears as well as by the sections, are specific to hydrophobia.

3. Numerous "bodies" are found in fixed virus.

4. "Bodies" are found before the beginning of visible symptoms, *i.e.* on the fourth day in fixed virus, on the seventh day in street virus, and evidence is given that they may be found early enough to account for the appearance of infectivity in the host tissues.

5. Forms similar in structure and staining qualities to the others, but just within the limits of visible structure at (1500 diam. magnification) have been seen. Such tiny forms, considering the evidence they give of plasticity, might be able to pass the coarser Berkefeld filters.

6. The "Negri bodies" are organisms belonging to the class Protozoa. The reasons for this conclusion are: (*a*) They have a definite, characteristic morphology; (*b*) this morphology is constantly cyclic, *i.e.* certain forms always predominate in certain stages of the disease, and a definite series of forms indicating growth and multiplication can be demonstrated; (*c*) the structure and staining qualities as shown, especially by the smear method of examination, resemble that of certain known Protozoa, notably of those belonging to the sub-order Microsporidia.

7. The proof that the "Negri bodies" are living organisms is sufficient proof that they are the cause of hydrophobia; a single variety of living organisms found in such large numbers in every case of a disease, and only in that disease, appearing at the time the host tissue becomes infective in regions that are infective, and increasing in these infective areas with the course of the disease can be no other, according to our present views, than the cause of that disease.

A somewhat similar rapid method of diagnosis is that given by Frothingham.[1]*

Cornwall[2] has a paper on recent advances of knowledge in connection with rabies, in which he points out that "Negri bodies" can be demonstrated in brains which have been ill-preserved and are even in a state of putridity. A microscopical diagnosis can now be made in a day or so. If "Negri bodies" are found, rabies can be safely diagnosed. If not found, and the specimen is fit for inoculation into a rabbit, the biological test can still be made, and in a few cases it succeeds where the "Negri bodies" have been missed by the microscope. It is evident that early diagnosis is important in the case of patients unwilling to go for treatment until the diagnosis is certain, while it is satisfactory for patients under treatment to know that the latter is absolutely necessary. While "Negri bodies" are easily found in the brains of animals dead from street virus, they are with difficulty found in fixed virus[3] brains, and then only in very minute forms. All observers agree that rabic virus filtered through a Berkefeld candle retains its virulence. The large "Negri bodies" cannot pass this filter, so if, as Negri holds, the brain is thickly studded with minute forms or spores, which are unstainable or ultra-microscopical, and, therefore, invisible, the filtrability of the virus is an argument in favour of the parasitic nature of these bodies.

Nitsch, while giving some very gratifying statistics as regards the Pasteur treatment in his hands, indicates further improvements in the method by combining injections of antirabic serum with injections of fixed virus, as Pasteur's method of immunisation can only succeed in those cases which have a sufficiently long incubation period to allow of immunisation before the outbreak of the disease.

Stefanescu[4]* has signalised the discovery of the "Negri bodies" in the salivary glands of mad dogs, while Babes[5]* believes: (1) That certain very fine spherical, black or blue bodies (Cajal-Giemsa stain) found in degenerated nerve cells represent the parasites of rabies in full activity; and (2) that the large "Negri bodies" are encapsuled forms in process of involution and transformation owing to the local reaction induced in the invaded cell.

Lentz[6] has recently described two new staining methods for the "Negri bodies," and illustrates the results by two coloured drawings which give a very clear idea of the form of the corpuscles.

Negri,[7]* continuing his previous work, indicates a cycle of development for the bodies bearing his name, which, though incomplete, is suggestive and strengthens the idea of their being protozoa. There are two phases: (*a*) large chromatin masses breaking up into

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[1] Frothingham, L. (April, 1906). *Journal of Medical Research*, Vol. XIV.

[2] Cornwall, J. W. (April, 1906), "Recent Advances of Knowledge in Connection with Rabies." *Indian Medical Gazette*, p. 121, Vol. XLII.

[3] "Fixed virus" results from the serial passage of "street rabies" through rabbits. It is exalted in virulence.—A. B.

[4] Stefanescu, E. S. (May 18th, 1907). *C. R. Soc. Biol.*, t. LXII.

[5] Babes, V. B., Press Medical (October 20th, 1906), *Zeit. f. Hyg.*, t. LVI., f. 3, May 24th, 1907.

[6] Lentz, O. (July 27th, 1907), "Ein Beitrag zur Färbung der Negrischen Körperchen." *Cent. für Bakt. I. Orig.* Bd. XLIV., p. 374.

[7] Negri, A. N. (May 5th, 1907). *Rendic d. R. Acc. dei. Lincei*, t. XVI.

* Article not consulted in the original.

(*b*) grains which are surrounded by a ring of protoplasm, and which are so small as only to be visible *en masse*. **Hydrophobia —continued**

Remlinger,[1] in an important paper, states that spontaneous recovery of experimental rabies can take place in the dog and that the rabic virus can persist in the saliva of recovered animals. The points he raises require confirmation, and are under the consideration of a special Committee.

Ice. A paper on the Pollution and Self-Purification of Ice, in the *Monthly Bulletin*, New York State Department of Health, of March, 1907, will amply repay perusal to all those responsible for the public health, and more especially in hot climates.

It is pointed out that there are very few cases on record where intestinal diseases have been traced to ice as the vehicle of infection and that, of these, one must clearly distinguish between intestinal disturbances caused by decomposing organic matter, and diseases, such as typhoid fever or cholera, caused by the specific germs. Some half-dozen supposed ice-borne epidemics, chiefly of enteric fever, are cited, and the conclusion come to is that the evidence is by no means reliable.

Ice may be infected because derived from polluted water, natural ice may be contaminated *in situ*, and either natural or "artificial" ice may be rendered impure during manufacture, handling or distribution. The last-mentioned is by far the most serious mode of infection, for it leaves the time interval, between infection and transmission, too short for any counteracting influence of cold to be exerted. The influences effecting self-purification of ice are:—

1. The phenomenon of subsidence and oxidation upon suspended matters, including bacteria in bodies of quiescent or slowly flowing water. This, of course, applies chiefly to ice from lakes and ponds. In rivers, however, it is found that in a run of from 10 to 15 miles, from 50 per cent. to 75 per cent. of the bacteria are removed by this means.

2. The removal of suspended and dissolved matters brought about during the freezing process, the so-called "filtration process of freezing." This in general effects a reduction of about 90 per cent. of the suspended particles and bacteria and also eliminates to a certain degree chemical compounds in solution.

3. And, most important, the effect of low temperature. The special point to note is that the period of exposure is of greater importance than the degree of cold. It is said that bacteria are reduced in number about 50 per cent. after exposure to freezing temperatures for one hour; 90 per cent. after 24 hours; and practically 100 per cent. after two to three weeks. The few killed after a month are so attenuated as to be unable to produce disease.

4. The general tendency to the destruction of pathogenic bacteria when exposed or disseminated through a body of water. This is no doubt due in part to unfavourable environment and in part to the hostile action of the common water bacteria. It is thus evident that in most instances it is the storage, handling and distribution of ice which require special attention from the standpoint of a health officer.

As regards Khartoum, a few remarks on this subject will be found under "Sanitary Notes" (Third Report).

Infectious Diseases. Under this somewhat comprehensive title we propose to consider only one point, namely, the early diagnosis of infectious diseases by recognition of the involvement of lymph glands.

Vipond[2] has a very interesting paper on the former subject, and his conclusions may be quoted with advantage. He says:—

1. The nodes are enlarged in infectious diseases.

2. They are enlarged some days before the development of the disease. I have found them to be enlarged and tender seven days before the rash of measles appeared.

3. The enlargement is most marked between the ages of from three to 18 years.

4. The enlargement is not produced by the irritation of the rash, but is due to the absorption of the poison or toxin.

5. As a rule nursing infants do not contract infectious diseases readily, as the tonsils are small and inactive.

6. The tendency to contract infectious disease would be much lessened if the mouth and tonsils were in a healthy condition.

7. The enlargement of the nodes is more marked in certain infectious diseases than in others. For instance, they are larger in erysipelas, measles and rubella than in scarlet fever and whooping-cough.

8. They resolve more quickly in diphtheria under the influence of antitoxin than they do in measles and erysipelas.

9. In all infectious diseases (except those of local incubation) the poison most likely enters the system through the tonsils.

10. Suppuration does not take place in the nodes unless we are dealing with a mixed infection.

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[1] Remlinger, P. (November, 1907), "Spontaneous Recovery of Experimental Rabies in the Dog and the Persistence of the Rabic Virus in the Saliva of Recovered Animals." *Journal of Tropical Veterinary Science*, p. 393

[2] Vipond, A. E. (December 15th, 1906), "The Early Diagnosis of Infectious Diseases by the Recognition of the General Involvement of the Lymphatic Glandular System." *British Medical Journal*, p. 1710, Vol. II.

Infectious Diseases— *continued*

Finally, when called to see a child suffering from an infectious disease, it is one's duty to examine the other children in the family who have been exposed, and, if the nodes are found to be enlarged, they too should be isolated immediately, and thus do away with the dangerous custom of billeting children upon friends and relatives, with the inevitable result of spreading the disease broadcast. I am satisfied that in the future the practice of isolating children already suffering from infected nodes will largely do away with epidemics in public schools.

A glance at the mortality tables of all our large cities will show a high death-rate from infectious diseases, and the early recognition of infectious diseases by means of the node involvement would result in these precautions, which would prevent the spread of the disease, thus materially reducing the death-rate resulting from infectious diseases.

Influenza. Influenza, or what is said to be influenza, although I do not know that any cases have been diagnosed bacteriologically, has occurred more than once in Khartoum, and doubtless true influenza is not uncommon in the Sudan, for the disease is by no means confined to countries with cold or temperate climates. Hence some very interesting and apparently none too well known facts brought out by Allbutt[1] may be mentioned with advantage. I have tabulated them just as they occur in the course of his lecture:—

1. Cases in which the respiratory tract is unaffected are not infectious, the disease being propagated in the sputum and spray from the respiratory tract and apparently in no other manner save, perhaps, at the very beginning of epidemics, for between epidemics the bacillus must be latent somewhere, unless, indeed, it is normally present in human beings, *i.e.* in the oral cavity, and for some reason or other takes on pathogenic properties.

2. The origin and habits of the parasite are still unknown.

3. Five to six months immunisation after attack is generally conferred even on very susceptible people. Twelve months is the more usual respite.

4. The inflammation appears to be of an erysipelatous type. There is a tendency to small cell and nuclear infiltration, there being far more intestinal infiltration than in croupous pneumonia.

5. The disease is very seldom confined to one lung.

6. Influenza, like phthisis, may excavate the lung.

7. In convalescence the malaise very often departs as suddenly as it came on, that is to say, a brusque recovery occurs in the course of a protracted convalescence.

8. Allbutt adds a fourth to those usually recognised, namely the continuous form. There is a persistent febrile state, though the temperatures are not high nor the symptoms severe.

9. In making the diagnosis, it is important to note that, unlike what is found in other febrile conditions, the urine in influenza is not high coloured and is not lateritious as in "chill." This would appear to be a practical point of very considerable importance.

10. Paraplegic attacks and peripheral neuritis may occur as sequelæ.

11. Angina pectoris may also be a sequel.

12. It is not safe to give a patient chloroform for some time after an influenzal attack, owing to the cardiac disability which ensues upon it.

Rogers[2] mentions that in India the type of the disease does not differ from that met with in Europe. The great frequency of respiratory and throat complications help to distinguish it from malaria and seven days' fever. Its diagnosis from dengue has been considered.

Williams,[3] following Corvisart and Wilks, speaks of the great value of an initial dose of opium in influenza. A full dose of 20 to 30 minims of liq. opii. sedativ. is given, and as a rule abolishes the distressing pains in a short space of time and ensures refreshing sleep. Indeed the action is so marked that it is probable the drug exercises a specific action on the Pfeiffer bacillus.

Nash[4] states that in every case of influenza there is a swelling and œdema of the uvula, which, as a result, has a pale, waxy, œdematous appearance. It is found at the commencement of the attack and usually lasts for several days. He regards it as a certain sign of the disease.

Insects. A good general article on the relation of insects to human diseases, albeit now a little out of date, will be found in "Harrington's Hygiene."[5] From this and other sources one has compiled a list of injurious insects and the human diseases they are known, or supposed, to transfer.

1. *Ants.* Cholera? dysentery? enteric fever? and indeed all those diseases due to contamination of food (*see* "Flies," *page* 75). There is no definite proof that ants act as vectors, but considering their habits in tropical countries, it is far from unlikely.

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[1] Allbutt, T. C. (May 6th, 1905), "Discussion on Influenza." *British Medical Journal*, p. 977, Vol. I.

[2] Rogers, L., "Fevers in the Tropics," 1908.

[3] Williams, W. L., "Minor Maladies."

[4] Nash, W. G. (April 4th, 1908). *Lancet*, p. 1032, Vol. I.

[5] Harrington, C., "Practical Hygiene." London, 1905.

Insects—*continued*

2. *Bed-bugs.* Anthrax ? cerebro-spinal meningitis ? endemic gastric catarrh of Austria-Hungary ? epidemic dropsy ? intermittent fever ? (Russia), leprosy ? Leishmaniosis ? mouse septicæmia ? plague ? skin diseases ? spirochætosis ? tuberculosis (Dewèvre's case), typhus fever, yaws ?

3. *Cockroaches.* Plague ?

4. *Fleas.* Miliary fever ? plague, tænia cucumerina, typhus fever ?

5. *Flies* (non-biting). Anthrax, conjunctival diseases, cholera, diarrhœa, dysentery, enteric fever, myiasis of various kinds, metazoan parasites (by transporting their eggs and depositing them on food) ; leprosy ? ophthalmia, skin diseases, staphylococcic infections, trypanosomiasis ? tuberculosis, yaws ? (Castellani).

6. *Flies* (biting, apart from mosquitoes). Anthrax (as in Cyprus). Septic and septicæmic infections, trypanosomiasis.

7. *Itch Insect.* Leprosy ? skin diseases.

8. *Lice.* Skin diseases. Spirochætosis.

9. *Mosquitoes.* Dengue ? distomiasis ? filariasis, leprosy ? malaria, Malta fever ? seven days' fever ? Weil's disease ? yellow fever.

10. *Ticks.* Black-water fever ? filariasis *(F. perstans)* ? plague ? (Skinner). Japanese River Fever ? skin diseases, spirochætosis, spotted fever of Rocky mountains ? trypanosomiasis ? (Manson's suggestion). Typhus fever ? yaws ?

One may also mention as of less importance certain Coleopterous and Lepidopterous larvæ which produce cutaneous irritation and inflammation, while spiders, scorpions and centipedes must also be kept in mind.

As regards the Bed-bug, the following is quoted from the *Indian Medical Gazette* of June, 1906 :—

There is a definite tradition in Russia, frequently repeated by Professor Metchnikoff, in his lectures at the Pasteur Institute, that the bed-bug forms an intermediate host or is at least an agent for conveying intermittent fever, so common in certain districts of that country. Its possibilities in the rôle of intermediary in cerebro-spinal meningitis are still the subject of investigation.

In these circumstances, all possible information with regard to the pest is interesting, and consequently a leaflet issued by the Department of Agriculture should attract attention. This pamphlet is written by Dr. Girault, who has carefully followed the life-history of cimex or clinocoris, as the insect is variously called in scientific nomenclature, and has found important new material with regard to its feeding habits. The adult insect feeds about once in from thirty-six to forty-eight hours, taking nearly fifteen minutes to get its fill of blood. At earlier ages, the feeding period is much shorter. Except in susceptible human beings who have a decided idiosyncrasy, no local effect at all is produced by the feeding process. Some individuals, however, suffer from almost intolerable itching and have a series of urticaria-like lesions. The parasite is so prevalent in certain parts of the country, however, that it is evident that those with an idiosyncrasy are comparatively rare. The insect may live from five to ten weeks or even longer without any food. This is especially true during the winter, when, if they have no food, they go into a short comatose condition. While each insect seems to live not longer than about 100 days at the outside, some have been known to exist as long as eight months; and it is these that enable the species to continue its existence even under unfavourable circumstances, so that they live from season to season in lumber camps, summer residences, empty apartments and the like.

Another interesting phase of investigation with regard to the bed-bug and its connection with disease was carried out by Drs. Girault and Strauss, as to whether or not the insect had other host relations besides those with the human race. As is easily understood, the host relations of this insect are greatly increased in importance because of their scope in the potential transmission of disease if they attack other animals than man. It has been found experimentally that at least mice, both living and dead, are attacked by bed-bugs and that young mice particularly seem to provide an excellent supply of food for them. This observation has been confirmed by observations made by Drs. Girault and Strauss, and, as it seems not unlikely that other small animals, including even rats, may also be hosts, the spread of such affections as pest, mouse septicæmia and the like by means of this parasite must be taken into account. In a word, a new departure of disease etiology seems to be opened up by these observations.

Cimex lectularius measures 4-5 mm. in length, 3 mm. in breadth, and has eight abdominal segments. The females deposit fifty whitish eggs at a time, three or four times a year, and cimex becomes mature in about eleven months. Bed-bugs live in cracks and fissures, under carpets, behind pictures, in furniture, bedsteads, etc. Hidden by day, at night they attack persons to suck their blood. An alkaline secretion of their salivary glands forms the so-called "wheal." Bed-bugs can migrate from house to house and may leave a house if it becomes uninhabited. The characteristic nasty odour of bugs is due to a clear, oily, volatile liquid secreted."

Patton deals with the distribution of the bed-bug, of which the two common species are *Cimex lectularius*, Linn., and *C. rotundatus*, Sign., as Patton has named *C. macrocephalus*. In a private communication, Patton informs me that the bed-bug from Angola is *C. rotundatus*, and suggests that it is probably the carrier of Leishmaniosis in the Sudan. The Khartoum bug is *C. lectularius*. Patton also points out that the bug lives on blood alone, not on moist

Insects— *continued*

wood, dust and dirt, as has been stated. His latest description[1] of bed-bugs may be quoted with advantage :—

The fact that the Leishman-Donovan body undergoes its extra-corporeal development in the bed-bug, *Cimex rotundatus*, makes it necessary to describe briefly the two species of this insect associated with man.

According to Mr. Distant, the family *Cimicidæ*, which contains four genera, *Cimex*, *Oeciacus*, *Cacodmus* and *Hematosiphon*, belongs to the *Heteroptera*, a sub-order of the *Rhynchota*, and is placed in the series *Gymnocerta* between the families *Phymatidæ* and *Certocombidæ*. Mr. Distant informs me the genus *Cimex* at present contains four species, *Cimex lectularius*, Linn., *Cimex rotundatus*, Sign., *Cimex pipistrelli*, Jenyns, and *Cimex columbarius*, all of which have the following characters: The head is short and broad with two prominent eyes, but no ocelli. The antennæ are four-jointed, the apical joints being slender; the elytra are rudimentary and lie over the metathorax. The prothorax is semilunar in shape with its anterior angles considerably extended. The abdomen, which is uncovered, consists of seven segments and an eighth anal appendage; the legs are slender, the anterior tibiæ more than twice as long, and the posterior three times as long, as the tarsi, which are three-jointed. The proboscis is flexed in a groove beneath the head and prothorax.

Type species, *Cimex lectularius*, Linn. The adult insect of this species is reddish-brown in colour and is covered with fine hairs; it varies in length from 4·5 mm. to 5 mm., the male usually being a little smaller than the female. The head is short and broad and is inserted into a notch in the prothorax; it contains two lateral prominent eyes in front of which are the two antennæ and in the median line the thick first segment of the labrum which is continuous with the dorsal integument of the head. The antennæ consist of four segments, the first two being the thicker, while the third and fourth are slender and covered with long hairs.

The prothorax is semilunar in shape with two rounded horns extending close up to the eyes; its upper surface is raised in the centre and towards the sides, ending abruptly at a line a little beyond the level of the eyes; the remainder of the surface, including the two horns, is flattened from above downwards. The ventral surface of the prothorax is concave and hollowed out on each side of the mid-line where the first pair of legs are inserted. The mesothorax, as seen from the dorsal surface, is triangular in shape with its apex projecting over the mesothorax and between the elytra. The mesothorax, next in size to the prothorax, is almost entirely covered on its dorsal side by the elytra, and on the ventral surface is seen as a small cleft on the inner side of the middle coxa. The elytra, which are inserted into the mesothorax just below the lateral angles, are two rudimentary scallop-shaped pieces of chitin lying over the metathorax and sides of the first abdominal segment. Their dorsal surfaces are convex and covered with bristles, while their ventral surfaces are concave. The abdomen is rounded and consists of seven segments with an eighth and appendage. It is broadest at the third segment and gradually becomes narrower towards the end where it is covered with long hairs. In the male, the penis is seen flexed in a notch between the seventh and eighth segments.

This bug is distributed through Europe and North America, and is also found in Suez, Egypt, the Sudan, the North-West Frontier Province of India, China, South Africa and Australia.

Cimex rotundatus, Sign., or the Indian bed-bug, is darker than the above species, being of deep mahogany colour; its head is not as long or as broad as that of *lectularius*. Its prothorax is also narrower and shorter, is more rounded and not flattened at the sides as is the prothorax of the type species. Its abdomen is orbicular, being broadest at the second segment, and tapers more abruptly towards the end; in all other respects it is similar to *Cimex lectularius*.

Cimex rotundatus is distributed throughout India, Burma, Assam, Malay, and is also found in Aden, Sierra Leone, the Islands of Mauritius, Réunion, St. Vincent and Porto Rico.

Cimex pipistrellli, Jenyns, is similar in colour to *rotundatus*, its prothorax is also less marginal, and in all respects it is more closely allied to the Indian bed-bug.

Cimex columbarius, Jenyns, has a similar prothorax to *lectularius* and is also of the same colour.

Dissection of the bed-bug. The following method of dissecting the bed-bug has been found satisfactory. After having killed the insect by placing it in a tube with a plug of cotton wool containing a few drops of chloroform, it is taken up with a pair of forceps and the legs are pulled off. In removing the fore legs it is necessary to be careful not to injure the prothorax, as they are firmly fixed in concavities on its under surface. The elytra are next removed by raising them with a fine pair of forceps and gently twisting them off from their joints at the angles of the mesothorax. The bug is now placed in a drop of normal saline solution with its head directed towards the dissector. A fine needle is inserted with the left hand into the right side of the prothorax, while with another fine needle in the right hand the joint between the prothorax and mesothorax is separated. By gently drawing the needle in the left hand and by exerting pressure on the dorsal surface of the abdomen with the other needle, the œsophagus, midgut and the remaining part of the intestinal tract are drawn out, and any portion can then be isolated and examined. The salivary glands lying on each side of the œsophagus are also exposed by this method. The ducts of the salivary glands and the œsophagus can be followed into the crop situated in the head by carefully removing the prothorax.

As the barracks in Khartoum used to swarm with bugs, and as they are troublesome in the quarters of the Public Works Department, these notes may be of some interest, though one has never heard of any similar condition prevailing locally.

The endemic gastric catarrh mentioned under "Bed-bugs" is described by Kirchenberger and Vala.[2]* It occurs during the summer months in the southern provinces of Austria-Hungary, and specially attacks soldiers in barracks. The incubation period is three to five days, followed by a prodromal stage of a few hours characterised by malaise, headache and

[1] Patton, W. S. (1907), "The Development of the Leishman-Donovan Parasite in Cimex Rotundatus." *Scientific Memoirs of the Government of India*, No. 31.

[2] Kirchenberger and Vala (September 3rd, 1907). *Allgem. Wien. Med. Zeitung.*

* Article not consulted in the original.

constipation. There is then a rise of temperature, the globes of the eyes become tender and there is a characteristic injection of the conjunctiva, hence the name "Dog disease" applied to the condition from the fancied resemblance of the affected eye to that of a dog. Mild bronchitis, gastric tenderness, cramps and epistaxis are the chief symptoms, together with a rash like urticaria or erythema multiforme. The disease lasts two to four days and terminates by crisis, but convalescence is slow and there may be considerable anæmia. A lasting immunity to further attacks seems to be conferred by the disease. It has been attributed to the bites of certain gnats but there seems more reason to believe that bed-bugs may be the vectors. **Insects—** *continued*

King[1] has described a very tiny blood-sucking *Hemipteron*, which he found in Khartoum. It attacks without provocation, but only causes slight local irritation.

As hornets are common in the Sudan, though apparently for the most part quite inoffensive, attention may be directed to a note wherein MacWatters[2] details three cases in which serious symptoms of collapse followed the sting of a hornet (*Vespa orientalis*).

Wellman[3] draws attention to the noxious larvæ of Coleoptera and Lepidoptera above mentioned, some of which may cause severe pain and skin eruption, while nervous symptoms may follow contact with stinging caterpillars. He has also a note on two species of Myriapods, and states that their poisonous secretion is probably from the foramina repugnatoria which are at the sides of the segments and look like trachael stigmata. One has had Myriapods sent from the Southern Sudan, some of which are said to be much dreaded by the natives. They are being determined by Professor Werner of Vienna.

Jaundice (*Infectious*). *See* Weil's Disease, *page* 231.

Kala-azar. *See* Leishmaniosis (*below*).

Leishmaniosis. Various reports have shown that this deadly disease is of much wider distribution than was at first thought to be the case. Assam, Bengal, Southern India, Ceylon, Burma, China, Arabia, Egypt, Sudan, Tunis, Algeria, South Africa and Crete are the places where it has been known to occur.

Most of the facts presented in various papers, together with original matter, are to be found in Rogers' work.[4] We can only quote some of the more important points to which he refers and supplement them from more recent papers.

In speaking of a certain epidemic, he explains the peculiarity of its course by the fact that the disease travelled through the virgin soils of a certain northern valley in Assam, previously unaffected by the sporadic form of the disease, and there found a population fully susceptible to its deadly influence. Hence it was able to work terrible havoc. This, if confirmed, is interesting in the light of a similar state of things probably existing in Central Africa as regards Sleeping Sickness, which invaded Uganda from the west with such dire results.

The early stages are very hard to diagnose, the differentiation from typhoid and paratyphoid being especially difficult. Rogers says the high continued type of fever, especially with a slow pulse, is almost conclusive evidence of typhoid as against early kala-azar, while the high remittent type is almost equally rare in the latter disease. Some typhoids, however, show the slow remittent type which is common in kala-azar, and if a negative serum test has also been obtained, the blood changes must be turned to for help.

Three of the most important early signs are:—

1. Double remittent type of pyrexia.
2. Persistent remittent fever with absence of severe constitutional disturbance and of abdominal or respiratory symptoms.
3. Enlargement of spleen down to the navel.

Rogers has shown that a marked relative leucopænia, say less than 1 white to about 1000 red corpuscles, is practically diagnostic of the disease and is of bad prognostic significance. There is nothing special to note as regards general symptoms, except possibly

[1] King, H. H. (December 15th, 1906), "Blood-Sucking Hemipteron." *Journal of Tropical Medicine*, p. 373, Vol. IX.

[2] MacWatters, R. C. (June, 1908), "Some Effects from Stinging by a Hornet (*Vespa Orientalis*)." *Indian Medical Gazette.*

[3] Wellman, F. C. (June 1st, 1907), "Notes on some Noxious Larvæ from Angola." *Journal of Tropical Medicine and Hygiene*, p. 185, Vol. X.

[4] Rogers, L., "Fever in the Tropics." (*Loc. cit.*).

Leishmaniosis— *continued*

that the majority of cases begin with rigors, but a good account is given of the changes in the spleen and liver. The former is usually hard and, in very chronic cases, its firm edge may project so as to be evident to sight through the abdominal wall. The rapid increase, and the still more striking rapid decrease, which may occur in the size of the spleen are described. In very chronic cases an actual cirrhosis of the liver may occur. The surface of the organ is smooth, and microscopically there is a very diffuse intra-cellular cirrhosis in the fibro-cellular tissue of which shrunken parasites of kala-azar may still be visible with a high power. Advanced cases are accompanied by ascites.

The chief blood change is a great relative reduction of the leucocytes which may be extreme even at a very early stage. There is usually also a marked increase in the percentage of the large mononuclears. This, be it noted, rarely occurs early in typhoid, and hence is a useful diagnostic aid.

Possibly improved technique will be able to demonstrate parasites in the peripheral blood even early in the disease. As regards the general course of the fever, many charts are given showing the different types of fever. A double remittent passing into a low fever is common, while the low continued type also occurs. Both high continued and the high remittent forms are much more rare.

Blood changes.

1. Marked anæmia is only characteristic of the later stages.

2. Relative leucopœnia is very marked and may be pathognomonic. It is less marked during high fever than during remissions or low intermittent pyrexia. It is important to note that a great degree of leucopœnia may be absent in kala-azar, (*a*) during any inflammatory complications such as pneumonia, dysentery, cancrumoris, meningitis, phthisis, etc.; (*b*) during high remittent pyrexia occasionally; (*c*) during the very earliest stages of the disease such as the first month of fever, or in recovering patients who have been free from fever for some time.

3. Increase in the large mononuclears. Note that kala-azar differs from malaria in that this increase seems to occur more frequently when there is high remittent fever than when it is intermittent or absent or when the temperature is normal. In malaria it is less marked or even absent during pyrexia. An increase of the large mononuclears in typhoid during fever is very rare, hence this sign is valuable in early kala-azar with high remittent fever which closely simulates that of enteric.

4. Decrease in the polynuclears. This, which is marked, is of significance in two directions. (*a*) As a prognostic sign, the prognosis becoming progressively worse as the polynuclears become fewer and fewer. (*b*) As a factor predisposing to the secondary inflammatory complications, often coccal or bacterial in origin, which so often prove fatal. This is easily understood when it is remembered that there may be a loss of nine-tenths of the phagocytic polynuclear leucocytes.

5. There is increase of the lymphocytes and decrease of the eosinophiles, but these changes are of no special import.

As regards treatment, Rogers upholds the utility of quinine given in large doses and for months together if necessary. He has repeatedly seen a high remittent fever reduced to a comparatively harmless low intermittent one by increasing the quantity of quinine given, say, up to 60 grains or even 90 grains a day. He also points out that a considerable number of cases wholly recover.

The parasite is then fully considered. It may be found in practically every organ of the body, but is most numerous in the spleen, bone-marrow and liver. Christophers' work is mentioned. It showed that the parasites multiplied mainly in the large endothelial or macrophagic cells of the spleen and bone-marrow, especially until the invaded cells bulge into the lumen of the vessels. Hence, when splenic puncture is performed, the larger capsulated forms are obtained.

It has been found that the parasite is absent from the body in diseases other than kala-azar.

The flagellated stage of the parasite is then discussed and its resemblance to Herpetomonas noted. These discoveries and observations are so well known that there is no need to refer to them here at any length. One important point, however, is the optimum temperature for the cultivation and development of the parasite. This is between 20° C. and 22° C. Hence, in working with bed-bugs, it is well to carry out the feeding experiments during the cold season. It was Rogers who determined that the reaction of the fluid in the stomach of the bed-bugs, after they had sucked human blood, was distinctly acid, and this led him to employ an acid medium (citrated human spleen blood plus sterile citric acid) for observing the development of the parasite. Prophylaxis on plantations and in villages in India is fully considered. Segregation of the sick, building of new lines and the destruction of old houses and purification of old sites by fire are advocated, as is the destruction of bed-bugs by sulphur fumigation, washing beds with strong boiling carbolic lotion, boiling clothes in the same or destroying them altogether and burning blankets.

Rogers,[1] in a recent paper, enters more fully into the question of the cirrhosis of the liver present in cases of kala-azar, and concludes that:—

1. The most chronic cases of kala-azar not infrequently terminate their course with ascites due to cirrhosis of the liver.

2. The cirrhosis is of a peculiar intralobular type of uniform distribution, and with a smooth surface to the organ.

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[1] Rogers, L. (July 1st, 1908), "A Peculiar Intralobular Cirrhosis of the Liver produced by the Protozoal Parasite of Kala-azar." *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. II, No. 3.

3. It is due to the protozoal parasite of kala-azar, which may be found in the liver and other organs after death. **Leishmaniosis—** *continued*

4. This form of cirrhosis of the liver is much commoner in Lower Bengal than a true malarial cirrhosis, with which it has probably hitherto been confused. It is, however, much less common than atrophic cirrhosis due to unknown causes.

Patton,[1] who has done the work on the development of the parasite in the bed-bug, records his latest work from which we cull a few notes and his conclusions. The large endothelial cells mentioned above, which contain the parasites in large numbers and are found in the liver, spleen, bone-marrow and lymphatic glands, occur in the peripheral blood of patients some days before death. "It seems most probable," says Patton, "that the rupture of these cells and the liberation of the parasites they contain, explains the occurrence of the large numbers of parasites in the peripheral blood of patients suffering from ulceration of the large intestine. Once the parasites are set free in the plasma they are immediately taken up by mononuclear, eosinophyl or polymorphonuclear cells."

It is suggested that the brawny swellings met with in the course of the disease, and *cancrum oris*, which is so common, both owe their origin to hæmorrhage caused by the blocking of a small vessel by one or more of these large cells.

Attention is drawn to large, non-nucleated, parasite-containing, blue-staining (with Giemsa) bodies, also found in the peripheral blood. Some believe them to be altered red blood corpuscles, others, detached buds of macrophages or mononuclear cells.

The general evidence seems to be against the occurrence of parasites in the red cells.

In continuation of his last report,[2] Patton gives more confirmation regarding the development of the parasites in *C. rotundatus*. After the early stages of development, *i.e.* increase in volume of protoplasm and in size of micronuclei which show signs of commencing division, one or other of two things takes place: (1) the parasite may rapidly pass on to flagellation; or (2), what is more important, they further enlarge and by consecutive division of the macro- and micronuclei produce a rosette of from four to eight parasites. "The single flagellates enlarge and begin to divide longitudinally to result in oval or spindle-shaped cells, while the rosettes, after flagellation, begin to divide up into separate, elongated flagellates. The oval or spindle-shaped parasites divide repeatedly . . . . and result in smaller and more irregular forms. All these changes may be seen in the bug during the first three days. Still later, longitudinally division of the oval and irregular flagellates progresses rapidly, so that by the fifth day the majority have become small or spirilla-like flagellates."

Patton also deals with temperature requirements and concludes that the disease usually begins in the cold weather, but this is not to say that infection can only take place during this season.

The final stages of development and the method of re-entry into the human body have not yet been determined, but the conclusions on this very valuable work so far are as follow:—

1. Though kala-azar is a chronic disease lasting many months and often years, it occasionally runs an acute course terminating in from four to five months, as illustrated by the case described above. In these acute cases, as well as in the chronic ones terminating with ulceration of the large intestine, the parasites are found a few days before death in large numbers in the peripheral blood in the leucocytes and endothelial cells, and the latter are probably the source of all the parasites seen in the circulating blood.

2. Though the parasites are most abundant in the peripheral blood towards the end of the disease, they are also found in the early stages, as a case recently seen clearly illustrated.

3. In the female as well as in the male bed-bug *(Cimex rotundatus)* the parasites have by the third day passed through all the intermediate stages of development described above up to the formation of the mature flagellates. Rapid multiplication by rosette formation is a "characteristic feature of the development of the parasite in the bed-bug." As the male bug sucks blood, it probably plays as important a rôle in the transmission of the disease as the female bug.

4. The infection acquired by the bug varies considerably, some ingesting large numbers of parasites, others only a few; and there is no evidence at present to show that the development in the bug depends on variations in the temperature.

5. The tendency that the disease has to linger in a house for a long time is probably explained by the fact that the parasite may remain in the midgut of the bug for several days before beginning to develop, and, as the nymphs, which take from seven to ten weeks to arrive at maturity, may ingest the parasites shortly after hatching, and as a rule feed only once between each moult, the infection may remain for a considerable time in a house; there is no evidence at present to support the view that the infection is inherited by the bug.

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[1] Patton, W. S. (1907), "The Development of the Leishman-Donovan Parasite in Cimex Rotundatus." *Scientific Memoirs of the Government of India*, No. 31.

[2] Patton, W. S. (1907), "Preliminary Report on the Development of the Leishman-Donovan Body in the Bed-Bug." *Scientific Memoirs of the Government of India*, No. 27.

Leishmaniosis— *continued*

Leishmaniosis was recently discussed[1] in London. Bassett-Smith referred to the occasional similarity of the temperature chart to that of Malta fever, the occurrence of hæmorrhages (epistaxis), the benefit resulting from atoxyl treatment, the drug being pushed to 12 grains a day, and the danger of splenic puncture. Liver puncture, he thought, was safe.

Precautions for puncture are: (1) Warn patient to avoid movement. (2) Thoroughly sterilise the skin. (3) Use a small needle and withdraw the blood quickly. (4) Apply a firm binder and make patient lie quite still in bed for several hours afterwards.

Manson described a case in a European which seemed to be about to terminate fatally and then markedly improved. This may or may not have been due to the atoxyl used in treatment.

Others spoke to the value of change of air and a sea voyage. The general impression was that when cure resulted, nature had been the successful physician.

Low mentioned a case clinically indistinguishable from kala-azar in which no parasites were found on spleen and liver puncture and in which, post mortem, double tumours of the adrenals were found, although there was no bronzing of the skin. He also mentioned other conditions, such as splenic anæmia, closely resembling kala-azar, and pointed out that for a certain diagnosis the parasite must be demonstrated.

Leishman mentioned Cummins' suggestion to produce artificial pustulation and thereby obtain polynuclears without having to resort to splenic or hepatic puncture.

Woolley[2] has described cases in the Philippines with symptoms of splenomegaly, rheumatic pains, œdema, diarrhœa, with or without hepatic enlargement, and remittent fever, but in which no Leishman-Donovan bodies could be found. He believes they may be associated with a chronic infection or intoxication originating in the intestinal tract and that, while a certain number of cases of tropical splenomegaly may be due to infection with the Leishman body, it will be found that various organisms are associated with the clinical picture and that the symptoms will depend chiefly on intestinal conditions and pathological changes in the intestinal walls.

Nicolle and Cassuto[3] have an interesting paper on three cases of splenomegaly in children at Tunis, in which cases the Leishman bodies were observed. There is a good coloured plate showing amongst other things a capillary of the liver with parasites in the endothelial cells and the forms met with in culture. The authors conclude:—

1. That there exists in Tunis an illness of infants with symptoms similar to those of kala-azar and apparently identical with certain cases of infantile splenic anæmia, described more especially in the south of Italy. It usually seems to terminate in death, but treatment by atoxyl may possibly prove beneficial. The etiology is unknown and the diagnosis can only be made with certainty by splenic puncture.

2. The pathogenic agent is a protozoan indistinguishable from the Leishman body. Its habitual site appears to be the endothelium of the vessels, and it is found in the blood and liver and more rarely in the lymphatic glands and in the blood.

3. This parasite is easily cultivated at 22° C. in the condensation water of blood-agar (Novy and McNeal's medium). The culture forms are identical with those of the Leishman parasite in citrated blood.

4. Until the identity of this parasite either with the Leishman body or the organism of oriental sore described by Wright, is established, it is proposed to group the following under the generic name Leishmania.

Leishmania wrighti, the cause of oriental sore.

Leishmania donovani, the cause of kala-azar.

Leishmania infantum, the cause of an infantile splenic infection closely resembling kala-azar, observed in Italy and in Tunis.

One's impression is, however, that the three cases recorded were merely examples of kala-azar affecting infants, and that there is no need to accord them special distinction.

In a later paper, Nicolle and Comte[4] mention the very interesting fact that they have found the parasite in a dog in Tunis. They also state that the children whom they found

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[1] "Kala-azar" (March 16th, 1908). *Journal of Tropical Medicine*, p. 85, Vol. XI.

[2] Woolley, P. G. (June, 1906), "Tropical Febrile Splenomegaly." *Philippine Journal of Science*, p. 533, Vol. I.

[3] Nicolle, C., and Cassuto, E. (February 1st, 1908), "Sur Trois Cas d'Infection Splénique Infantile à Corps de Leishman observés en Tunisie." *Archive de l'Institut Pasteur de Tunis*, p. 3.

[4] Nicolle, C., and Comte, C. (April 11th, 1908), "Origine Canine du Kala-azar." *Archive de l'Institut Pasteur de Tunis*, p. 59.

infected had been in habitual contact with dogs. They are inclined to think that infection may be derived from the dog, and that in this animal the disease runs a benign course. Their work, which is of a preliminary nature, requires confirmation, but is at least suggestive. Leishmaniosis—*continued*

The presence of Leishmaniosis in the Sudan was first determined by Neave, who reported a case in a child from Meshra-El-Rek. For a considerable period thereafter no further cases were recorded, but recently, thanks to the work of Captains Cummins and Bousfield, of the Egyptian Medical Service, a considerable number of instances of the disease have been brought to light. In addition, as already mentioned, Dr. McTier Pirrie fell a victim to it, and I have found the parasites in the splenic smears of several cases sent me for diagnosis from Wad Medani. A second fatal case in a European has recently occurred.

As Captain Cummins furnishes a special paper on this important subject there is no need to enlarge upon it here.

Its distribution has yet to be fully worked out, but it seems commonest in the Kassala and Sennar Provinces, at least at the present time. So far *C. lectularius* is the only species of bug found in the Sudan, but it is possible that *C. rotundatus* will be discovered in the regions where kala-azar is endemic.

I have little doubt but that the disease is much more common than is generally supposed and that hitherto it has been overlooked or not recognised. Every effort will have to be made to arrest its spread should it show signs of extending beyond its present known limits.

Leprosy. In looking over the recent literature relating to leprosy, one notes the tendency to attribute the disease in the first instance to the bites of insects.

Thus Mugliston[1] of Penang suggests that *Acarus scabiei* may be the carrier of infection. He found that of 77 lepers, 44 had itch on admission, 11 of the others remembered having itch, and from the remaining 22 no history was obtainable. He thinks it possible that the bacillus of leprosy may be conveyed either *in* or *on* the insect.

Bassewitz[2]* also cites a case where in all probability leprosy was conveyed by the parasite of scabies from a leper to his attendant. The incubation period was two and a half years.

Goodhue[3] of the Leper Settlement at Molokai, Hawaii, reported the finding of the bacillus of leprosy both in the female mosquito, *Culex pungens*, and in the bed-bug, *Cimex lectularius*. He is inclined to think that the bug is a greater factor than the gnat in the spread of leprosy.

Smyth[4] of Lebombo states that in his diocese there are tribes which till quite recently have been free from European influence. The cases of leprosy amongst them are sporadic. These natives eat and drink out of the same pots; if there be food, no matter of what kind, they all share alike. They practically live altogether, with one exception—no healthy person ever sleeps with a leper, or one suspected of leprosy. In the case of married people, they have intercourse, but it is always in the daytime if one is a leper. The children of such parents appear to be normal. The sick are isolated in a hut or huts quite close to the village, but no healthy man or woman sleeps there. Smyth argues with some force that if the intermediate host were winged, it would fly from hut to hut, and therefore leprosy would spread in the villages in a way that it does not seem to do; if it were infectious, like some other diseases, people would catch it from the common spoons and cups; if it were due to food, all who eat that food would be liable to get it. Therefore we are driven to the conclusion that the *Cimex lectularius*, or some similar wingless parasite that feeds at night, is the means of conveying it from man to man.

Hutchinson[5] adduces the following arguments against the insect theory:—

1. If such conveyance were possible, how account for the disappearance of the disease in the fifteenth and sixteenth centuries from England and from Europe?

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[1] Mugliston, T. C. (July 15th, 1905), "On a Possible Mode of Communication of Leprosy." *Journal of Tropical Medicine*, p. 209, Vol. VIII.

[2] Bassewitz, E., *Münch. Med. Woch.*, 1905, No. 41.

[3] Goodhue, E. S. (August, 1906), "The Bacillus Lepræ in the Gnat and Bed-Bug." *Indian Medical Gazette*, p. 342, Vol. XLI.

[4] Smyth, W. R. (December 8th, 1906), "Leprosy." *British Medical Journal*, p. 1670, Vol. II.

[5] Hutchinson, J. (December 22nd, 1906), "Mosquitoes and Leprosy." *British Medical Journal*, p. 1841, Vol. II.

* Article not consulted in the original.

Leprosy— *continued*

2. Leprosy now prevails extensively in Southern China and is unknown, for the most part, in the North.

3. Husband and wife scarcely ever suffer together.

4. The medical officers and nurses of leper asylums never contract the disease.

5. In all leper asylums there are a certain number of inmates who have been admitted under erroneous diagnosis. These often remain there for years, yet they never acquire leprosy.

6. The lepers who are brought from abroad into England and Northern Europe never become sources of infection to others. Further, it does not seem probable that the prick of an insect should convey a disease respecting which carefully-conducted experimental inoculation always fails. Moreover, leprosy never shows any primary sore.

Turning to other aspects of the question, an analysis of 220 cases of Sudanese leprosy by Tonkin[1] is of special interest to us here. The Central Sudan, *i.e.* the region about Sokoto, Kano and Kuka is that concerned. In many ways the habits and customs of the races inhabiting this part of Africa appear to be identical with those of the people of the Anglo-Egyptian Sudan, and to anyone anxious to work out the leprosy problem in the latter country, a perusal of Tonkin's paper is indispensable. In a summary he shows that the age of onset seems to work out earlier for the Sudan than in some other exploited areas, but he notes that this may be due to some special local conditions which have escaped attention. He says little about recovery from the disease, but indicates that in the Central Sudan as elsewhere there is evidence to show that it occurs and that this question is one meriting more attention that it has yet deserved.

The history of his cases does not support the factor of heredity as being instrumental in the production of the disease and he brings forward arguments against it, for example: leprosy is a disease which reacts prejudicially on propagation, hence it is not likely to derive support from a factor that is hereditary. As regards predisposition, he shows that leprosy has occurred in many people who have had no leprous ancestors. He believes deficiencies of diet have a great deal to do with it, coupled with faulty hygienic principles as regards clothes and bedding. He says that wherever leprosy occurs we have habits with regard to clothes and bedding which are open to criticism and stereotyped national diets which leave much to be desired. He remarks that in the Central Sudan the communistic way in which unwashed clothes are handed about must have a direct influence on the spread of leprosy, and that a diet in which the carbohydrate elements are represented to an extent which disturbs the proper proportion of the nitrogenous would appear to handicap races reared on it in their fight against the disease. He does not, however, believe that an ill-assorted diet will cause leprosy or that the disease cannot be contracted without the intervention of such a diet. Still he does maintain that in the Central Sudan the most frequently operating factor, not in causing the disease but in assisting to determine its incidence, is that of a badly balanced and, therefore, inefficient diet.

It is worth noting that fish is recorded to be dear and, therefore, consumed by the masses of the country even to a less extent than meat, which is by no means a staple form of food. Salt throughout the whole country is scarce and dear.

These latter paragraphs lead us into the realms of controversy, and though it is, of course, impossible to quote fully from the literature which has accumulated regarding the fish theory of leprosy so ably and ingeniously put forward and tenaciously supported by Jonathan Hutchinson, reference must be made to several of the more interesting recent papers available.

The whole subject is considered in a review[2] of Hutchinson's work "On Leprosy and Fish Eating: A Statement of Facts and Explanations." Hutchinson's theory is that the fundamental cause of true leprosy is the eating of fish in a state of commencing decomposition. The cause of the disease is, in his opinion, some ingredient or parasite generated by, or introduced into, fish which has been either not cured at all or cured badly. As a result, it is maintained that segregation of lepers will not stay the disease.

So long as the food is not taken from the hands of lepers it is asserted that there is no danger from consorting with them.

The various arguments brought forward to prove the fish theory are detailed at some length, such as the irregularity of the distribution of leprosy and the fact that a very large number of lepers are found in all countries who have neither inherited the taint nor been in any way exposed to contagion.

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[1] Tonkin, T. J. (April 18th, 1903), "An Analysis of 220 Cases of Sudanese Leprosy." *Lancet*, p. 1077, Vol. I.

[2] Hutchinson, J. (June 16th, 1906), "On Leprosy and Fish Eating: A Statement of Facts and Explanations." Review in *Lancet*, p. 1695, Vol. I.

Leprosy—*continued*

The conditions under which the disease has declined in certain countries and has held its ground in other countries are cited, New Zealand, Spain and Crete being examples of the latter state of things. In Norway, leprosy is limited to fishermen, boatmen and peasants, whose staple food consists of milk and the products of the sea. Peasants inland, who suffer, have free access to fish food during its conveyance from the sea.

As the majority of observers do not accept Hutchinson's deductions, the arguments against them in this review may be given in full :—

It must be confessed that this interesting theory does not meet with the approval of a large number of physicians who have had personal knowledge of the disease. Let us note some of the objections to it. And first as to bad fish being the essential factor. Should any cases be shown to exist where bad fish has not been the essential factor, it must follow that the theory falls to the ground. Are there such cases? We have seen that the author explains the decline of leprosy amongst the Maoris to the fact that they have changed their diet to one of meat, potatoes and bread. Now Dr. Francis Day, whilst travelling in Burmah, never saw a case of leprosy amongst the indigenous population, although the Burmese like fish, and especially the odoriferous "nga" fish. Dr. Vincent Richards, a man of acknowledged authority on tropical subjects, shows by statistics not only that the consumption of fish has nothing to do with leprosy as regards its causation, but he also states that it would be quite as logical to state that this factor is a prophylactic, the disease being less met with in those districts where there is the greater consumption of fish ; and lastly, Dr. Crane, the Government physician in Hawaii, has written that from time immemorial fish, especially raw fish, had been the staple food there until the year 1860. Until that year there were no lepers in Hawaii nor was there any history of the disease. At this date it appeared, and in a few years after its introduction it had decimated the community, yet at this time canned foods, fresh meat, and vegetables had been very largely substituted for fish in the diet, and the use of fish, especially raw fish, had enormously decreased. The history of Hawaii, in fact, demonstrates this fact, "all fish, no leprosy," "little fish, no leprosy," and the argument is as valid as that used by the author in the contrary sense, concerning the Maoris. Again, experience in India demonstrates the fact to the physicians in that country that many lepers declare, not only that they have never eaten fish, but that they actually do not know what it is. Caste ordinances, again, militate against the use of fish in many who have become lepers.

Let us consider, also, the question of contagion. How can the fish theory explain the cases shown by Dr. Benson at the Dublin Medical Society in 1872 and 1877? In 1872, a leper was shown by him who had acquired the disease in the West Indies. In 1877, Dr. Benson showed a second case in the person of the brother of the first, who had never been out of the United Kingdom. This man had slept in the same bed with his brother and occasionally worn his clothes. Consider, again, the case mentioned by Professor W. Osler. Here seven people were in turn affected by what to most minds must seem to have been contagion. And in connection with the question of contagion, the remarks of Von Duhring are peculiarly apposite—namely, that all negative evidence brought forward as to its non-communicability is valueless in the face of one positive fact. Lastly, as regards the alleged failure of segregation to prevent the disease, few of those who have had personal experience will agree with the author. To mention one instance only, in Madagascar the former limited amount of leprosy has rapidly increased since segregation was abandoned. It is thus seen that there are many objections to Mr. Hutchinson's theory which must be overcome before it can be finally accepted as the true explanation of the disease.

Indian experience is also adverse, as evidenced by another review[1] which we quote at some length, partly because the arguments used appear to be very sound, and partly because one has been specially asked to furnish information on this subject as regards the prevalence of leprosy in certain parts of Kordofan where there are lakes containing fish.

After pointing out that the important question of personal immunity is wholly ignored in Hutchinson's work, the reviewer says :—

Passing now to the consideration of the fish hypothesis itself, we find that the chief reasons for which fish is considered to be the article of food responsible for the propagation of leprosy are these. Granting that it is due to an article of food, it must be one which is of universal use and which has the same quality in all lands. There is no vegetable met with in all the districts affected by leprosy; the vehicle is not milk, for some races, the Tartars for instance, almost live on milk and yet have no leprosy, and there are many leprous centres where milk is almost or wholly unknown; while of flesh foods there is no reason to suspect that of animals and birds. We may note that the author has not taken into account the most widespread article of food, namely, salt. All places where leprosy now prevails (he continues) are either on the sea-coast or near to rivers or lakes, except in the case of India, and this exception he considers as probably not so great as it at first sight appears. The disease is of such special and emphatic individuality that it can have but one and the same cause. That cause is in nineteen-twentieths of its instances fish, and respecting the twentieth he considers that despite defects in evidence the inference is justified that it is fish. But, we point out, the cause is, in all cases, the bacillus, irrespective of its vehicle or point of introduction, and the same bacillus will produce the same reaction in the human being in whatever way it may gain entrance to the tissues. As regards the kind of fish which is considered to cause the disease, one finds from scattered references up and down the book that although salt fish is constantly spoken of as a cause of leprosy, this is not what is really meant. Properly salted fish is looked upon as perfectly safe. The article which is considered to be the cause of leprosy is fish in an early stage of decomposition, either from not having been preserved at all, or from having been merely dried or improperly salted and subsequently eaten uncooked. We believe that in one place only in the book (*page* 49) is it stated that it is uncooked fish of the character just stated which is considered responsible for the transmission of leprosy, and although much space is devoted to the attempt to prove that leprosy is associated with the ingestion of decomposing fish, no attempt at all is made to show that the fish is uncooked or imperfectly cooked. Having followed the argument so far, the conviction occurs that here, at all events, is something which is capable of proof or disproof, seeing that all

[1] Hutchinson, J. (August, 1906), "On Leprosy and Fish Eating." Review in *Indian Medical Gazette*, p. 327, Vol. XLI.

Leprosy— *continued*

that is necessary to test the matter is to ascertain whether the incidence of leprosy is co-extensive with the use as a food of decomposing fish. Such an expectation is doomed to disappointment. We read: "The fish hypothesis assumes that it is probable that even in fish in a state of decomposition the presence of the dangerous ingredient is exceedingly rare, but that a very small quantity of fish containing it may be efficient in the production of the disease. Thus the large or small consumption of fish has little to do with the matter." In other words, Mr. Hutchinson has hedged, and the hypothesis as now enunciated by him is incapable of proof or disproof, and his challenge of refutation he can make with perfect safety. The whole tenor of the book shows that the author occupies towards his offspring the position of an interested advocate, and never that of an impartial judge. The fish hypothesis is supported thus: As a general statement it is true that all places where leprosy is now prevalent are either on the sea-coast or near to rivers or lakes; all over the world it is a disease of tribes which fish and not of those which hunt. It was very prevalent in Europe in the middle ages and began to decline before the reformation, the conclusion reached being that its prevalence was due to the eating of improperly dried fish on fast days, and its disappearance to the religious laxity which he assumes to have preceded the reformation. He shows quite clearly that at that time isolation of lepers was not attempted. Leprosy has not been prevalent in Russia because the Greek Church does not allow of the eating of fish on fast days. It occurs more in men than in women, partly because men no doubt get an unfairly large share of the decomposing fish food where it is an expensive luxury, and partly because men are dirtier feeders. Among the Hawaians the appearance of leprosy, forty years after white men came among them, was coincident with the establishment of a factory for curing fish. From Cape Town dried fish is distributed largely to the Malmesbury district, a noted centre for lepra. Leprosy in Norway only occurs on a strip of the western coast where there is little land for cultivation, and fish is the principal article of food and is preferred tainted. There is a large export of dried fish from Norway to Spain and from Newfoundland to Portugal, and there is much leprosy in Spain chiefly along the north and south coasts. We should imagine that were there any value in this argument, the incidence of leprosy in Spain and Portugal would follow the trade routes, and not be chiefly incident on the more inaccessible north and south coasts.

The large proportion of lepers in India who are Christians, he considers a proof of his hypothesis. We have ascertained from the manager of a leper asylum with 200 inmates that nearly all the Christian lepers there have become Christian since their admission into the asylum as lepers. There is one part of Mr. Hutchinson's argument, which we do consider to be built on a basis solid and capable of being upheld, though we entirely dissent from the conclusion drawn from the facts; we refer to the inverse ratio between the abundance of salt and the incidence of leprosy. The author gives the amount of salt required daily by an adult as from 300 to 400 grains, including that present in the food; he states that the development of the salt trade and the introduction of better kinds of salt have often been coincident with the decline of leprosy in a very marked manner, that for example one great difference between Northern and Southern China is that in the one salt is abundantly produced, and is an article of export, while in the other it is imported and subject to tax, that in the latter leprosy is abundant and in the former unknown. He brings forward other statements of a like nature. Almost the whole of the salt used for fish-curing in Norway is imported, and we presume therefore dear, and that it is unlikely that the poor will get it in sufficient quantities. It is worthy of note that it is precisely those parts of Europe which are the most remote from the beaten tracks of commerce, and to which the importation of salt in common with other commodities would be unsatisfactory, that leprosy has lingered longest. Doubtless no one predisposing cause accounts for all the facts. In some places, probably the Sandwich Islands group is one of them, leprosy has appeared recently because it has been recently introduced, and just as measles, having within comparatively recent years been introduced into Iceland, the Faroe Islands and the Fiji Archipelago, has raged virulently among hereditary unprotected races, so has leprosy in the Sandwich Islands increased by leaps and bounds, till 40 years after its introduction, every fiftieth individual was a leper. Some, too, of the world-wide decline of leprosy may probably be fairly put down to the natural decline of an epidemic disease in the course of years, one of the most familiar examples of which is plague."

Jennings[1]* notes that in Abyssinia there are some 8000 lepers, who can rarely, if ever, have eaten fish, owing to the scarcity of the article in a country where, "during the greater part of the year, many of the wells do not contain water, much less fish."

An interesting paper on leprosy in Cape Colony is that by Black.[2] He also argues against the fish theory as regards the Cape, and draws special attention to his observation that the disease begins (how long after the implantation of the bacilli cannot at present with certainty be known) as a small collection of round cells under the mucosa of some part of the nasal cavity. Some of these contain bacilli which pursue some part, at any rate, of their life-history in their interior. Their dissemination to distant organs appears to be mainly by the blood stream, and these cells come to rest in situations where there is a tendency to intermittent stasis or slowing in the fine capillary circulation. Hence the lungs, brain and spinal cord, being furnished with a rapid, free and continuous current of blood are practically exempt from the occurrence in them of leprotic granulomas. On the other hand, just as we have in leucocythæmic conditions an infiltration and enlargement of the liver and spleen, so we have such in leprosy by bacilli-containing leucocytes.

In connection with the nasal-infection it is noted that one of the earliest signs of nodular leprosy is a slight erythema of the skin about the root of the nose and eyebrows, sometimes extending to the malar regions, and that with these slight signs extensive

[1] Jennings, J. W., "With the Abyssinians in Somaliland." Quoted in *Journal of Tropical Medicine*, February 15th, 1908, p. 62.

[2] Black, R. S. (April 28th, 1906), "Remarks on Leprosy in Cape Colony." *Lancet*, p. 1167, Vol. I.

* Article not consulted in the original.

ulceration of the nasal mucous membrane may be found with abundant bacilli practically in almost pure culture. **Leprosy—** *continue*

Nasal obstruction is often the earliest symptom, though naturally its significance is frequently unrecognised. Black admits that great difficulties surround the actual circumstances of the communication of the disease from individuals, and, from the fact that it seems almost impossible to reproduce the disease in animals, concludes that there is some very special relation between seed and soil. He thinks that the agency of an intermediate host, be it flea, bug, or acarus has not been proved, and mentions that if the nasal mucous membrane is the primary seat of infection then the intermediate host theory, however enticing, must go by the board.

In a later paper, Black[1] again deals with the question and advances a theory to account for the marked clinical difference between maculo-anæsthetic leprosy and nodular leprosy. He believes that nodular and mixed leprosy are simply maculo-anæsthetic leprosy plus the infiltration and œdema of the subcutaneous tissue in various parts of the body, caused by the active invasion of the *Bacillus lepræ*. Continuing, he says:—

In practically all cases of nodular leprosy anæsthetic and leucodermic patches can be found just as in maculo-anæsthetic leprosy. I also noticed that when a nose had fallen in, in some cases of nodular or mixed leprosy the type of the disease in the individual seemed to change and he became practically, in course of time, a maculo-anæsthetic case, the infiltrations being gradually absorbed. I further noticed that some cases of undoubted maculo-anæsthetic leprosy were of an extremely mild character. A patient would have a few anæsthetic patches on his body with, perhaps, a contraction of the small and ring fingers of one of his hands and would otherwise be in perfect health—in which condition he would remain for years. I also found that practically all the active cases of mixed and nodular leprosy suffered from rhinitis and were discharging bacilli with the nasal secretion in great numbers, whereas, except in a few of the early cases, I found that the maculo-anæsthetic patients had no bacilli in their nasal secretion. The above considerations afforded me what I consider to be a solution of the problem. I think that there can be hardly any doubt that leprosy in its early stages begins as a small ulcer on some part of the extensive nasal mucous membrane. We know quite well, from our clinical experience of the disease, that leprous ulcers in favourable circumstances tend to heal. There can, therefore, be little doubt that a person can suffer from a leprous ulcer in the nose that may heal and pass entirely away. This is the explanation of the maculo-anæsthetic cases. They have had nasal ulceration which has passed away, in some cases leaving perhaps a cicatricial shrinking of the nasal septum, but during the time the ulcer existed leucocytes or white connective tissue corpuscles got detached from the ulcerating spot and, along with the bacilli which they were attempting to devour, were carried by the blood stream and lodged in various situations in the peripheral nerves, where they got entangled, and the bacilli then proceeded to grow, causing pressure on the fine nerve fibrils and consequently setting up nutritive changes in the skin which these axis-cylinders supplied, thus causing the patches of discoloration and anæsthesia.

In the nodular and mixed cases the progress of the disease is quite different. Instead of the nasal ulcer healing up it proceeds to grow apace, producing extensive destruction of the nasal mucous membrane, causing it to swell up and ultimately attacking the nasal bones themselves. We have something to guide us from what we know of other diseases affecting the nose, such as fleshy growths from the nasal mucous membrane, and how, if not treated, the nose becomes distended and the aspect of the patient altered. Also in the disease called post-nasal adenoids in children, how from their intra-nasal pressure they cause the face to assume a heavy, puffy and stupefied appearance. A similar condition takes place in nodular leprosy. The accumulating swelling of the mucous membrane extending into the numerous interstices of the naso-pharyngeal cavity causes the face to assume its characteristic appearance of this clinical variety of the disease, assisted of course by the leprotic infiltration from the festering nasal ulcer. But the festering sore has other consequences. The enormous number of leucocytes which are on the scene trying to overcome the invading bacilli get many of them translated to other parts of the body. They carry with them bacilli which in leprosy seem to have the property, at any rate at some part of their life-history, of growing within the white blood corpuscles and thus propagating the disease through all the parts of the body. As in maculo-anæsthetic leprosy, they get entangled in the peripheral nerves and cause anæsthesias and the discolorations of the skin and they also in nodular leprosy form large accumulations in the liver, the spleen, and in certain situations in the subcutaneous tissues throughout the body, and the patient becomes at last, if not properly treated, a mass of ulcerating sores.

The course of the disease is, as is well-known, extremely protracted, and the cases which we see in our asylums are in nearly every instance in a very late stage of the disease, even on admission. The very early stage of the nasal ulceration, naturally not being recognised, is completely overlooked, even by the patients themselves. They think that they are perhaps merely suffering from a protracted nasal catarrh, to which they pay no attention and which they forget all about when the graver symptoms of the disease make their appearance. But at these extremely early stages of the disease, these people are sources of infection, and this fact affords a very clear and simple explanation of those mystifying and apparently hopelessly inexplicable cases of leprosy which from time to time occur. You ask one of these patients: "Has he ever been in contact with a person suffering from leprosy?" And he vehemently and perfectly truthfully affirms that he has never been in such contact. But though he did not know it, and though the communicator of the disease did not know it, he has been in contact with a person suffering from the primary early rhinitis of leprosy.

This view, as the author remarks, brings leprosy, as far as pathology and treatment are concerned, into line with tuberculosis and diphtheria, and he makes the suggestion that possibly the opsonic treatment of Wright might be applied with advantage.

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[1] Black, R. S. (October 20th, 1906), "A New Aspect of the Pathology and Treatment of Leprosy." *Lancet*, p. 1064, Vol. II.

Leprosy— *continued*

Hillis[1] refers to Black's work on the nasal conditions which he, to some extent, anticipated, but mentions non-tuberculated or anæsthetic cases of leprosy with reliable histories in which from beginning to end there was no nasal affection whatever.

Clift[2] suggests that leprosy, like tuberculosis, may have an intestinal origin. Thus flies may infect fish and other food-stuffs which under certain conditions would form a good nidus for the bacillus. The benefit resulting from taking salt may, he thinks, be explained by the resulting increase of hydrochloric acid helping the digestive juices to destroy the germs.

The blood in leprosy has recently been the subject of study by Bourret.[3] He examined ninety cases in French Guinea and found changes which, though marked, cannot be considered as characteristic. The hæmoglobin value is lowered, the number of red corpuscles diminished and the erythrocytes frequently present morphological lesions (anisocytosis, vacuoles, poikilocytosis, polychromatophilia, granular basophilia), while normoblasts also occur. The absolute number of leucocytes is generally diminished, never increased. In all the forms of the malady, whether or not attributable to it, there frequently exists an eosinophilia which may be considerable. The relative proportion of the lymphocytes is often augmented, while the large mononuclears remain almost constantly below the normal. Often enough there is an eosinophile and neutrophile myelocythemia, but slight in extent.

Passing now to the question of treatment, the *Lancet*[4] notes that for many years preparations of mangrove, *Rhizophora mangle*, have enjoyed a great reputation as a cure for leprosy amongst the people of Cuba, and goes on to record the results obtained by Duque and Moreno, which were certainly very remarkable.

A fuller account, dealing also with other work by Padilla in Central America, is quoted by Nicholson[5] as follows :—

> The effects of mangrove on leprosy are as follows. At first, if administered in large doses to commence with, it produces nausea, lassitude, vomiting and purging, which, however, disappear with the suspension of the treatment. For this reason it is always advisable to commence with small doses, in order to gradually accustom the patient to the drug.
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> The first thing the patient experiences after 15 to 20 days' treatment is an unaccountable feeling of cheerfulness, apparently without cause for same, and without doubt he has a better appetite, and the lepralgic pains are not so severe; he feels active and disposed to carry on his usual occupation. The following month the patient is another man; he has put on flesh, eats well, digests well, sleeps well and works without feeling weary; the spots have become somewhat rose-coloured, more so in the periphery than in the centre, and those which are confluent tend to separate, leaving large intervals of healthy skin between each other; if there are any ulcerations suppuration is diminished in them, they lose their dark colour and take on a bright red hue, and the inflammatory areola disappears, the lepromata undergo a transformation, the fresh nodules are not so extensive, and the fever usually present on these occasions almost entirely disappears. Following up the treatment, the improvement becomes more marked, the periodic crops of lepromata disappear definitely from the fifth to the seventh month of treatment, and the lepro-tuberculomata disappear in two ways, either by becoming inflamed, painful and suppurating, or else they undergo fatty degeneration and become absorbed, leaving behind them in each case a white scar with a faint, rose-coloured tint. Sometimes they become encysted and calcified, necessitating their removed by incision; the ulcers about the eighth month of treatment have healed up. The anæsthesia has completely disappeared. At the ninth month, according to Drs. Duque and Moreno, the alopecia ceases, and the eyelashes and eyebrows grow again. What is most noticeable in these patients is the return of the face to its normal condition, for it would at first seem impossible that faces with a skin like parchment, which give this characteristic and repulsive aspect, could return in a relatively short time to their normal condition with a fine skin. In a year, according to these same Cuban physicians, the patient is generally cured.
>
> Dr. Padilla now gives an account of three cases which came under his own personal observation. The first of these patients was under treatment for ten months, the second one for seven months, and the third for barely six months, and he states that the first and third cases gave the best possible results; but in the case of the second patient it was noticed that scarcely any hairy down reappeared on his body, nor did his eyelashes and eyebrows make much progress as regards growth. The first case occurred in a man aged 32 years, a widower, and a field labourer, with no family history whatever of the infirmity from which he suffered, which he believed he contracted from a female servant who lived in his house. His two sons, one aged 11 years and the other 9 years, are both healthy.
>
> He stated that the first manifestations that he noticed occurred about six years ago, commencing in the left cheek, and about two months later in the right cheek, and these were like thick ridges which increased until they covered the whole of his face, and were accompanied with painful pustules which afterwards disappeared. A year

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[1] Hillis, J. D. (December 1st, 1906), "A New Aspect of the Pathology and Treatment of Leprosy." *Lancet*, p. 1544, Vol. II.

[2] Clift, H. L. (April 24th, 1907), "Intestinal Origin of Leprosy." *British Medical Journal*, p. 931.

[3] Bourret, B. G. (January 22nd, 1908). *Bull. de la Soc. de Pathologie Exotique*, t. I, No. 1.

[4] *Lancet* (October 21st, 1905), "Mangrove Bark in Leprosy," p. 1201.

[5] Nicholson, J. E. (October 2nd, 1905), "Treatment of Leprosy by Mangrove." *Journal of Tropical Medicine*, p. 293, Vol. VIII.

Leprosy—*continued*

afterwards he had more spots, which spread to the nose and ears, taking on a blackish tint with scales which had disfigured his nose, and some ulcerated nodules on his ears, and also scattered over many parts of his body, including his nails, and certain affected parts were also attacked by local inflammation. Dr. Padilla diagnosed the case as one of tubercular leprosy in its third stage, and he commenced treatment by 30 grammes of mangrove boiled in 120 of water, to be taken daily; this was done for twelve days. For another similar period the dose was increased to 45 grammes daily, and then for another ten days to 60 grammes a day. After this the fluid extract was used, commencing with 20 grammes daily, the dose being gradually increased up to 45 grammes in a month's time; a great improvement was now observed, including the reappearance of downy hair, the swelling of the feet disappeared, the patient enjoyed a good appetite and slept well, was very cheerful, and was now able to attend to his usual occupations. The fluid extract was now changed for the dry in doses of 2 grammes daily, which were gradually increased up to 7 grammes in the next two and a half months. Eleven months after the commencement of the treatment the patient was the first to ask for its suspension, since he felt and appeared to be thoroughly restored to health.

The treatment has also been tried in the Havana leper hospital during more than two years, on 17 cases, the drug being given internally and applied locally, and though the results obtained were not equal to those recorded by Duque and Moreno, considerable improvement occurred in every case, one being completely cured with disappearance of Hansen's bacilli.

Diesing[1]* reports cures by means of subcutaneous injections of iodoform. He employs a 30 per cent. emulsion of iodoform in olive oil and injects from 2 c.c. to 8 c.c. of this daily under the skin, at first in the neighbourhood of the infected areas, and later, when the lesions are healing, in some suitable situation. The dose is increased fairly rapidly to 8 c.c. and the desired result is obtained in about six weeks. Tincture of iodine may also, with advantage, be painted on externally. It is suggested that either the amount of iodine liberated is sufficient to disinfect the whole body, or that the bacilli, having been killed by the iodine, produce or set free antibodies, which eventually attack and destroy all the organisms present in the body.

Thompson[2] reports a case in Australia which failed to benefit by the administration of gurjun oil, but was greatly improved, indeed was very nearly cured by chaulmoogra oil *(Oleum gynocardium)* and frequent hot baths. The dose of oil at first was 45 minims a day given in three doses. This was finally increased to 270 minims and for a time was combined with strychnine. A good deal of benefit, however, may have been due to causes inherent in the patient. In the account of the case, Thompson mentions two special methods of examination which can be employed to test whether leprosy is or is not present. These we quote as likely to be useful, and they are said not to be generally employed:—

(a) The way is systematic examination of the peripheral nerve trunks as a matter of routine. In the accounts referred to these are most often omitted from mention; and if their condition be noted, then it is done only by way of adding one additional item to the complex of signs and symptoms from which the diagnosis must be deduced. But I now submit that such alterations as have been mentioned above, however (hardness, insensitiveness, increase in size, etc.) suffice by themselves to suggest a diagnosis of lepra when they are exhibited in a person who otherwise is in apparent good health. In declared cases, such alterations are always to be found if they are looked for; also they occur very early, and I doubt whether they may not be the earliest sign of infection.

(b) The means referred to is the subcutaneous injection of a salt of pilocarpine. I think that the appearance under the influence of pilocarpine of more or less dry areas of skin, intermingled with areas over which sweating occurs in normal degree, always affords strong ground for suspecting leprosy, while dryness of the wrists and hands and of the ankles and feet, when it occurs under that influence, is decisive.

Neumann[3]* combined chaulmoogra oil with salol and iothion. If not well borne by the stomach resort may be had to subcutaneous injection. This may rarely induce pulmonary embolism (Tourtoulis Bey), but it is a transient affair and disappears spontaneously.

A suggestive lecture on leprosy is that by Thompson,[4] in which he alludes to several interesting points:—

1. The lepra bacillus is not found in all of those lesions which, clinically and pathologically, denote the leprous process; thus it has been found absent from nerves which were markedly involved, and also from other lesions, despite numerous and careful examination by competent observers. "Hence," says Thompson, "while we have for the present no classified proof that the *Bacillus lepra* is the *cause causans* of leprosy, occasionally cases are met with clinically in which, according to common experience, the bacillus should be easily demonstrable, and in which, nevertheless, it cannot be found."

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[1] Diesing. *Berl. Klin. Woch.*, June 7th, 1906.

[2] Thompson, J. A. (December 1st, 1906), "Lepra Tuberosa—Treatment with Chaulmoogra Oil." *Lancet*, p. 1506, Vol. II.

[3] Neumann. Quoted in *Medical Annual*, 1907, p. 353.

[4] Thompson, J. A. (November 30th, 1907), "Notes on Leprosy." *Lancet*, p. 1514, Vol. II.

* Article not consulted in the original.

Leprosy— *continued*

2. Supposing it be the cause, it would seem that every leper, or at least every leper who discharges and scatters leprosy bacilli, must be a danger to others. But this does not follow; and observed facts show that some third factor is indispensable. Indeed by Dr. Thompson's arguments one is reminded of the X, Y and Z of Pettenkofer as regards cholera.

3. Observations in New South Wales have shown that avoidance of association with any leper is not enough to avoid attack (*see*, however, Black's paper, previously quoted, on concealed nasal infection), while on the other hand close association with lepers is a matter of very little practical importance at most. Indeed the evidence available makes it more probable that the infection of leprosy is received by man from his environment rather than from the sick.

4. It is quite possible, nay, even probable that infection may be taken by ingestion, and this despite the work of Woit and others who are inclined to believe that the infection is taken up by the skin and is essentially as ascending neuritis which leads ultimately to generalised infection of the body. Facts against this view are given, the most important being that in cases of almost universal leprous œdema, tactile pressure, heat and pain sensations sometimes appear to be perfectly normal, even after four or five years of manifest disease.

5. Evidence can be advanced against the hypothesis that the infection is commonly received by the nasal mucous membrane. Cases are not uncommon in which no evidence of such affection can be got. Thompson's experience is that the nasal lesions supervene earlier or later in the course of illness, and only according to the patient's inaccurate observation seem sometimes to have preceded the obvious signs of a generalised infection.

6. The successful inoculation of apes by Nicolle,[1] if confirmed, may yield an increase in our knowledge.

7. After further, none too favourable, comment on the chaulmoogra oil treatment, the "nastin" treatment, introduced by Deycke Pasha and Raschad Bey, is mentioned, "nastin" being a substance extracted from a special organism found in leprous tissues.

Recently full details of this treatment, as modified by Deycke, have been published.[2] The organism in question is not the lepra bacillus which could not be cultivated, but a form of streptothrix which has been termed—*Streptothrix leproides.* "Nastin" is a fatty principle extracted from this organism. When injected into lepers it gives rise to a reaction similar to that produced by tuberculin in tuberculosis. Now it was found that benzoyl chloride, even in the cold, deprives tubercle bacilli of fat in about twenty minutes, and does so instantaneously and completely after slightly heating. (Tubercle bacilli were used in the preliminary, experimental work, as they could be cultivated). The treatment briefly stated, is to employ benzoyl chloride and "nastin" together. As Deycke says:—

> Numerous trials having demonstrated that pure benzoyl chloride solutions are quite ineffective specifically, we can only assume that benzoyl is the really active substance which acts direct on the lepra exciter; it requires, however, in order to get at the bacillus at all and not to be wasted in other directions, the guidance, so to say, of the otherwise thoroughly passive "nastin," which charges itself in oily solution with benzoyl chloride; it is not, however, a chemical union but rather a state of simple solution; in this way benzoyl chloride is protected from being prematurely or uselessly used up. The "nastin" is carried to the lepra bacilli, to which, owing to its near chemical and physical relation, it attaches itself, and then benzoyl can fully display its anti-bacterial action in the fat-removing sense. When deprived of fat the lepra bacilli seem to be doomed; the human organism then effects with comparative ease the further dissolution and ultimately the complete destruction of the bacteria nuclei. Benzoyl-nastin, then, is an agent which directly acts on lepra bacilli. When administered in appropriate doses it brings about, if not in every case complete cure, at least the arrest of the disease in all but the most hopeless. The benzoyl-nastin solution is put up in ampoules containing each from 0·0005 to 0·002[3] nastin in sterilised olive oil. One such dose is injected hypodermically once a week. No reaction is observed with the smaller doses, but with the larger reaction occasionally takes place. It is not desirable to induce a marked reaction. In favourable cases, after some weeks or months the lesions gradually disappear. Kolle & Co., Biebrich am Rhein, have undertaken to prepare the material in a form ready for use.

It must be evident from these notes that an investigation on leprosy as it occurs in the Sudan might well repay the observer and furnish useful information. It is one of those diseases about which the last word is far from having been spoken, and, both as regards etiology and treatment, remains in an unsatisfactory condition. My own experience of leprosy is too slight to justify any comments upon a disease, which, while not very common, is by no means rare in the Sudan. It is said that there are between 30 and 40 known cases in Omdurman, but, as far as I can determine, the disease is more prevalent in the Kassala Province and in certain parts of Kordofan.

As a rule, nothing is done to control it, but cases are noted, and in Khartoum the disease is notifiable. It is very seldom, however, that we hear of a case.

In the light of recent work and observations it is questionable, apart from the benefit which patients derive, if leper hospitals do much good, at least as regards preventing the spread of the disease, in a country like the Sudan.

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[1] Nicolle, C. (May 15th, 1906), "Recherches Experimentales sur la Lèpre." *Annals de l'Institut Pasteur*, p. 389, Vol. XX.

[2] Deycke Pasha (April 4th, 1908), "A Lecture on a Specific Treatment of Leprosy." *British Medical Journal*, p. 802.

[3] Presumably of a gramme.—A. B.

Through the kindness of Colonel Hunter, I have had access to a very valuable and suggestive report, by Captain R. G. Anderson, on Leprosy in Kordofan. He shows that it is almost entirely limited to the Gebel district in the southern part of the Province, and that, as an endemic disease, it seems to exist in no other part of the Province. The anæsthetic type is by far the most common, cases of nodular leprosy being rare. Mixed cases are encountered. The patients are usually between 20 and 30 years of age; but, unhappily, children are not infrequently victims, and these suffer from the rapid, nodular variety and consequently undergo mutilation from the disease. Captain Anderson believes that close personal contact is an important factor in spreading the disease, and testifies to the influence of dirt and insanitary surroundings. Thus amongst the nomad, cattle-owning Bagara Arabs the disease appears to be unknown, probably on account of their cleanly habits, the care with which they anoint their bodies with oil, their healthy open-air life, and their simple diet of milk, bread and butter. **Leprosy**—*continued*

Of special interest are Captain Anderson's observations on the relation of leprosy in Kordofan to the practice of eating imperfectly cured fish. In a paper in the *Journal of Tropical Medicine* for April 15th, 1904, I mentioned the presence of mud fish in certain lakes in Kordofan, and stated that it would be interesting to know if these fish served as food and if leprosy occurred in the neighbourhood. Captain Anderson shows that there is a large fish industry, that the fish are greatly prized as articles of diet, being at the dry season of the year dug out of the mud in which they bury themselves. These fish are very badly cured, and are eaten when in an advanced state of decomposition, and it appears certain that there is a relative and close distribution of leprosy throughout the localities where fish eating is in vogue, while the disease does not exist where there is no fish traffic. Captain Anderson's investigations strongly support Hutchinson's fish theory, so far as Kordofan is concerned. His paper also includes proposals for isolation, segregation and general sanitary measures, and is undoubtedly the most valuable contribution that has been made to our knowledge of leprosy in the Sudan.

A dried fish food is sold in the market at Khartoum. It is, however, well preserved and is so desiccated that it bears no resemblance to the flesh of fish. The presence of bones and fragments of cartilage testifies to its origin. It is largely consumed by natives, and seems to produce no ill effects.

Liver Abscess. On the ground that prevention is better than cure, Rogers'[1] work on the "Pre-suppurative Stage of Amœbic Hepatitis: its Early Diagnosis and Cure," claims first attention. First of all allusion is made to the prolonged fever which so often precedes suppuration in the liver and which is commonly diagnosed and treated as malaria, and to the frequent necessity, even in the presence of acute hepatitis, for exploratory puncture in order to determine if an abscess has formed. Reference is made to earlier work on the value of leucocytosis in acute hepatitis, which, if marked, points to suppuration having taken place, but which in slighter degree may be present in the absence of pus and may indicate an early and curable stage of amœbic hepatitis. This view has been further developed, and as the author says:—

> These cases constitute a distinct class of fever, usually of a chronic intermittent type, sometimes with no very definite symptoms of hepatitis, and rarely with any dysentery. They may be recognised, or at least strongly suspected, by the presence of a moderate degree of leucocytosis, generally of the type which I have described as common in amœbic abscess of the liver, namely, one in which the proportion of polynuclears is either normal or only slightly in excess. Further, and this is the most important practical point—this kind of fever yields rapidly to large doses of ipecacuanha in the absence of symptoms of dysentery, or even of hepatitis, and the formation of tropical abscess of the liver is thus prevented.

Notes and charts of 15 cases follow. The blood counts and differential leucocyte counts are given. The polynuclears ranged from 74 to 87 per cent., the lymphocytes from 7 to 22 per cent., the mononuclears from 3 to 7 per cent., and the eosinophiles from 0 to 4 per cent.

In only three cases was over 80 per cent. of polynuclears observed, hence the type of leucocytosis present is similar to that which obtains in cases of amœbic abscess as noted above. The effect of the ipecacuanha treatment certainly seems to have been both immediate and remarkable and there can be no doubt but that it should be tried in all cases prior to any operation. Rogers believes this pre-suppurative condition with its attendant fever and apparently characteristic blood findings is due to the presence of a latent form of dysentery, the amœbæ of which are causing an irritation of the liver, which organ they are reaching mainly by the portal circulation. Ipecacuanha is to be regarded as a specific in amœbic dysentery, hence it is easy to understand how large doses of the drug (not less

[1] Rogers, L., "Fevers in the Tropics." See also *Indian Medical Gazette*, September, 1907.

Liver Abscess— *continued*

than 20 to 40 grains once or twice a day, with the usual precautions), may rapidly abort an early pre-suppurative amœbic hepatitis by curing the latent amœbic dysentery which produces it.

Stannus[1] has recently reported an African case similar to those described by Rogers. It occurred in Nyasaland and was rapidly cured by ipecacuanha given in 20 grain doses on two consecutive days.

Rogers and Wilson[2] describe two cases of amœbic abscess of liver cured by aspiration and injection of quinine into the cavity without drainage. In the first place, Rogers mentions his previous work,[3] which demonstrated that:—

1. Living amœbæ can be found in the walls of every case of tropical abscess of the liver examined at the time of or shortly after operation, as well as post mortem, if they have not been previously drained for some days.

2. Two-thirds of tropical abscesses of the liver seen in Calcutta are free from staphylococci and bacteria when first opened, even in the late stage of the disease so commonly met with in native patients, while, as a matter of fact, in nearly half of the remaining cases, but one or two colonies of cocci were obtained on culture, which were probably accidental contaminations or at least played no part in the causation of the disease.

3. The active amœbæ in the wall of a liver abscess could be killed with great rapidity and certainty by weak solutions of quinine, and when such solutions are used for washing out liver abscess cavities which are discharging thick pus swarming with amœbæ, these parasites very rapidly disappear, and the discharge becomes much less copious and almost serous in character, as long as it remains free from septic cocci.

The form of quinine recommended is the soluble bi-hydrochlorate. Two solutions are made up, each containing 30 grains, but in one this amount is dissolved in 2 oz. of water and in the other in 4 oz. The former is used if the abscess contain less than 10 oz. of pus, and the latter if it be larger, because the greater quantity of fluid enables the drug to be brought into contact with all parts of the cavity. It is admitted that two cases do not furnish sufficient material on which to base any conclusions, but the cases were of such a nature that it is very unlikely that aspiration alone would have resulted in cure. The first case was very acute, the second very chronic.

Cantlie[4] deals with liver abscess from the surgeon's point of view. After stating that supra-hepatic abscesses seldom give a history of dysentery, nor show bowel lesions post mortem, that intra-hepatic abscesses are always associated with, and probably caused by, dysentery, and that sub-hepatic abscesses are not due to dysentery, he proceeds to indicate what in his opinion is the appropriate operation for pus in various positions. He says:—

The operation I prefer (*a*) when pus is deep-seated over or in the right half of the liver, is evacuation by siphonage, the siphon tube being introduced through a cannula. Search is made first by the needle of an aspirating syringe—in length not more that 3¼ in., so as to avoid wounding the inferior vena cava. Where the pus is localised a large trocar and cannula is introduced trans-thoracically; the trocar is withdrawn, and a drainage tube, 12 in. long, stretched on a long steel rod, introduced through the cannula to the bottom of the cavity. The cannula is then withdrawn over the tube whilst it is still stretched, the steel rod by which it is stretched is also withdrawn, and a long rubber tube fitted to the drainage tube protruding from the side by means of a short glass tube. The siphonage is kept up until the pus ceases to flow, or until bile appears in the discharge. The original tube is then removed from the side, and a shorter and smaller tube substituted. The wound is gradually allowed to close. (*b*) When the pus is superficial—that is, close beneath the right ribs, an advanced condition, a hepatic abscess should never be allowed to attain—the trocar and cannula and siphonage may be used, or the abscess may be cut down upon by a scalpel. The removal of a piece of rib may or may not be necessary. If the knife happen to enter the lowest point of the abscess cavity, removal of a piece of rib is unnecessary; but, if not, it is well to do so. (*c*) When the pus is in the left half of the liver—a rather rare occurrence—do not attempt to confirm the diagnosis by introducing an exploratory needle, nor use the trocar or cannula, but cut down upon the liver through the abdominal wall and evacuate the pus in the usual way. In 90 of the 100 cases I have operated upon I employed the trocar and cannula and siphonage method of treatment; and from a long experience I look upon this method of treating deep-seated liver abscesses of the right half of the organ as not only the most easy of performance (an important point if one is single-handed, as one is in many tropical countries) and most successful, but the only justifiable operation when the pus is deep seated in the right side, that is, three or more inches from the surface.

Bradshaw[5] relates an interesting case of tropical liver abscess associated with ascites, a rare condition and one which tends to confuse the diagnosis. He mentions certain points which, though well known, are apt to be forgotten. One is that a normal temperature may be present for days together in patients with abscess of the liver. Another, that one

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[1] Stannus, H. S. (July 4th, 1908), "A Note on Latent Dysentery in Central Africa." *Lancet*, Vol. II.

[2] Rogers, L., and Wilson, R. (June 16th, 1906), "Two Cases of Amœbic Abscess of Liver." *British Medical Journal*, p. 1397, Vol. I.

[3] *British Medical Journal*, September 20th, 1902.

[4] Cantlie, J. (November 9th, 1907), "One Hundred Cases of Liver Abscess." *British Medical Journal*, p. 1342, Vol. II.

[5] Bradshaw, T. R. (January 18th, 1908), "A Clinical Lecture on Tropical Abscess of the Liver." *Lancet*, p. 146, Vol. I.

Liver Abscess—*continued*

of the chief difficulties in diagnosis is when liver abscess stimulates disease of the right lung or pleura or when it is complicated with effusion into the pleura. Pleural infection readily occurs, infective matter spreading by way of the lymphatics.

Bousfield[1] has recorded from Kassala a case of liver abscess, in the pus from which, taken soon after operation, a diplococcus was found simulating the gonococcus in appearance and staining reaction.

Liver abscess is by no means uncommon in the Sudan. I am unable to give an opinion as regards its relationship to dysentery, but I have recorded a case[2] of some interest in which the *Entamœba dysenteriæ* was found. I know now, however, that I was wrong in attributing the fatal termination of this case to shock. It was undoubtedly in the main an instance of delayed chloroform poisoning dependent on the state of the liver, and I only make mention of it here as a warning regarding routine administration of chloroform in cases of operation for liver abscess, or at least such administration combined with the usual preliminary starvation.

Reference to this important matter will be found in a paper by Stiles and McDonald,[3] which gives the bibliography, and in an article by Hunter,[4] who states that evil effects may in all probability be completely prevented if, instead of withholding food, particular care be taken that the patient be given a very nutritious and easily digestible meal, well sweetened, two or three hours before the operation.

Malaria. It seems advisable to classify the various papers for review as far as possible, though some dealing with several or many aspects of the disease cannot be placed in any one group. First then we may consider papers relating to the morphology or life-cycle of the parasite :—

A question to which a good deal of attention has been directed, both by Ewing[5] and by Craig,[6] is that of so-called intra-corpuscular conjugation. While, so far as I can ascertain, Craig's views on the subject have not been generally accepted, and while Cropper[7] has recently shown that when double or treble infection occurs each parasite seems to go on to full development (he found three præsegmenting forms in one cell), still Craig's latest paper on latent and recurrent malarial infection is both able and interesting, and it seems worth while to record some of his opinions. His definitions may be given :—

By latent malarial infection is meant one in which the plasmodia of malaria may be demonstrated to be present in the blood of an individual, but in which no clinical symptoms of the disease of sufficient gravity to attract attention are to be observed. The term should not be confined to those instances in which no symptoms of malaria have ever been present, for if the parasites be present in the blood in recurrent cases, between the attacks, the disease is as truly latent as it may be before the initial one.

By recurrences are meant the appearance of symptoms due to the same group of parasites that caused the original infection and not a re-infection by another group.

By intra-corpuscular conjugation is meant the complete and permanent union of the protoplasm and nucleus of two young amebula *(sic)* within the erythrocyte. It is absolutely necessary to the maintenance of malarial infection in man, and in these instances in which it does not occur, the plasmodia undergo a sexual sporulation for a limited time and then perish, thus leading to spontaneous recovery. It is present most typically in those cases in which the clinical symptoms are most severe, and is present in all the varieties of malarial infection, although most easily observed in the estivo-autumnal infections.

His conclusions regarding its significance are as follows :—

1. Intra-corpuscular conjugation is the chief cause of the maintenance of malarial infection.

2. It maintains malarial infection by producing a resting, or *zygote*, stage of the plasmodia, within the human body, which is resistant to quinine and other injurious influences.

3. It is the cause of latency and recurrences of malarial infection, the *zygote* stage remaining dormant or "latent" until conditions are favourable, when it gives birth to several young plasmodia, thus causing a recurrence of the infection.

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[1] Bousfield, L. (January, 1908), "A Case of Liver Abscess due to a Diplococcus Similar in Appearance and Staining Reaction to the Gonococcus." *Journal of the Royal Army Medical Corps*, p. 80, Vol. X., No. 1.

[2] Balfour, A. (November 21st, 1903), "A Case of Multiple Liver Abscess." *Lancet*, p. 1425, Vol. II.

[3] Stiles, H. J., and McDonald, S. (August, 1904), "Delayed Chloroform Poisoning." *Scottish Medical and Surgical Journal*, Vol. XV., No. 2.

[4] Hunter, W. (April 4th, 1908), "Delayed Chloroform Poisoning: Its Nature and Prevention." *Lancet*, p. 993, Vol. I.

[5] Ewing, J. (1904), "Clinical Pathology of the Blood."

[6] Craig, C. F. (June, 1906), "Observations upon Malaria: Latent Infection in Natives of the Philippine Islands—Intra-corpuscular Conjugation." *Philippine Journal of Science*, p. 525, Vol. I., and (Jan. 1st, 1907) *Journal of Infectious Diseases*, Chicago.

[7] Cropper, J. (March 16, 1908), "Phenomenal Abundance of Parasites in the Peripheral Circulation of a Fatal Case of Pernicious Malaria." *Journal of Tropical Medicine and Hygiene*, p. 91.

Malaria— *continued*

These conditions are justified by the following considerations:—

1. The presence of the process in all acute and recurrent infections.

2. The fact that during the time consumed in conjugation in all other protozoa, provided the conjugation is asexual, many generations of the organism could have been produced by division or sporulation in the usual manner.

3. The fact that such a resting, or *zygote*, stage must exist, as proven by the recurrence of the infection after the discontinuance of quinine given for long periods of time.

4. The fact that, in cases which have been treated at once with sufficient doses of quinine and for a sufficiently long period, intra-corpuscular conjugation is never seen, and in such cases relapses are very rare, if they occur.

5. The presence of numerous large pigmented bodies in the blood in cases in which the process is most marked, both intra- and extra-cellular, and which are not seen in cases in which the process is absent.

6. The argument from "analogy" which indicates that the significance of the process of asexual conjugation in the malarial plasmodia is similar to the same process in other of the protozoa.

In the Sudan I have seen one case which certainly suggested an intra-corpuscular conjugation, but there is no proof that it really does occur. Is it possible that the malarial parasites in the Western Hemisphere differ in certain respects from those of the Old World? It is not at all likely, and yet it is curious that it seems to be only from America and the Philippines that reports concerning this condition emanate. Ewing, it should be mentioned, regards intra-corpuscular conjugation as of rare occurrence and of comparatively little significance.

The Sergents have described a vermicular, endoglobular form of the malarial parasite seen in the blood of a native of Algeria, and Billet,[1] commenting on this form, mentions a gregarine stage previously studied by him,[2]* and states that the most favourable time for observing the perfect hæmogregarine form is at the beginning of the apyrexial period. Billet[3] has also described curved, vermicular forms of the quartan parasite assumed by the young schizonts during the first hours of their development. These have less active movements than those of the corresponding form of the tertian parasite and they do not throw out prolongations as do these latter. They eventually develop into the quadrilateral nearly adult schizonts characteristic of the quartan parasite.

In the blood of a case which had become infected at Taufikia, on the White Nile, I found the curious amœboid forms shown in Plate VII, Third Report. A parasite closely resembling a trypanosome will be observed. It differs from the hæmogregarine forms described by Billet, and, as it was the first parasite found in the film, proved, for the moment, puzzling, although clinically the case was one of malaria. On the following day a few crescents were found. Quinine soon caused the disappearance of the endoglobular forms.

Plehn[4]* records a case of tropical malaria acquired in Togoland, which, afterwards in Germany, following treatment, became a double benign tertian. The probability of the patient having had a latent benign tertian is negatived by the excessive rarity of such in the district from which he came. It would seem that a single species of malarial parasite is able to undergo variations according to the different countries and climates in which it develops. Ziemann,[5]* however, states that all three forms of malarial parasite occur in tropical Africa, and that all that can be said is that the benign tertian parasite is rarer in the Cameroons (1·1 per cent.) than in equatorial Africa, where Koch found it in 10 per cent. of the cases. Plehn's supposition is, therefore, probably faulty.

One can only note a paper by Billet[6] on the specific differences between the tertian and quartan parasites, and record his opinion that the quartan fever attacks certain races. Hence he believes there are morphological, clinical and ethnological differences between these forms of the malarial parasite.

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[1] Billet, A. (April 15th, 1905), *C. R. Soc. Biol.*, t. LVIII.

[2] Billet, A. (June 10th, 1901), *Ac. Sc.*

[3] Billet, A. (1906), *C. R. Soc. Biol.*, p. 1146, t. LX.

[4] Plehn, A. (July 25th, 1907), "Zur Frage der Arteneinheit des Malariaparasiten." *Deutsch Med. Woch.*

[5] Ziemann, H. (November 14th, 1907), "Zur Frage der Arteneinheit des Malariaparasiten Bemerkung zu dem Artikel von Prof. A. Plehn." *Deutsch Med. Woch.*

[6] Billet, A. (March 15th, 1908), "Preuves en Faveur de la Destruction Specifique des Hematozoaires de la Fièvre Tierce et de la Fièvre Quarte." Quoted in *Bull. de l'Institut Pasteur*, p. 194, t. VI.

* Article not consulted in the original.

Thiroux,[1] on the other hand, supports Laveran's view as to the unity of the malaria parasite. He examined native children in Senegal and found that in the hot weather the tropical forms amounted to 9·5 per cent., and large forms (benign tertian and quartan) to 1·5 per cent., while in November and December the respective figures were 73·5 and 26·4 per cent., and in March and April they were 64·1 and 35·8 per cent. He considers it difficult to admit the existence of a summer and winter malaria due to absolutely different species. **Malaria—** *continued*

It appears to me that some work on these lines could be carried out with advantage in the Southern Sudan, and possibly one may be able to arrange for this in the next programme drawn up for whoever may have charge of our Floating Laboratory.

Cropper[2] exhibited very interesting blood-films from a fatal case of pernicious malaria which contained a phenomenal abundance of parasites in the peripheral circulation. His description of the slides shown may be quoted:—

1 and 2. Clumping of infected red cells, each containing a pigmented præsegmenting body, suggesting a cause for the embolism or infarction of the different organs affected in pernicious attacks.

3. Very numerous unpigmented rings; of 500 corpuscles counted at random from 40 to 50 per cent. were infected, and in some fields more, so that in one field 100 parasites can be seen. Up to this time twenty parasites in a well-spread field was the most I had seen.

4 and 5. A red cell containing six unpigmented rings, in another slide; two corpuscles can be seen in one field, each containing five rings.

6. *Subtertian gametes or crescents* in all stages of development.

Towards the end the number of crescents increased in a marked manner, four, five, or six were seen in a single field, and suggest to my mind some sort of migration from the bone-marrow on the onset of algide symptoms. I give this for what it is worth. In very few cases are the crescents mature, and they are nearly always straight and fusiform, not crescentic; the pigment is nearly always discrete. The young male crescents are often perfectly circular in outline, and occupy the centre of the cell, being surrounded by a ring of hæmoglobin, the female gametes being sharply pointed. They can be distinguished from young segmenting forms by the fact that the pigment in the latter is nearly always gathered into a compact mass, even at an early stage. In any case, the chromatin exists as a distinct band across the Equator.

7. *Two sporulating bodies in one cell,*

8. *Three præsegmenting forms in one cell.* These, though somewhat rare, tend to show that when double or treble infection occurs, each parasite goes on to full development, and effectually precludes the occurrence of any form of endocorpuscular conjugation, as described by Craig, of the American Army, in the Philippines.

9. *Polynorphonuclear leucocyte* containing sporulating body.

10. *Leucocyte containing three sporulating bodies.*

11. *Pigmented leucocyte containing twenty-five pigment masses,* each indicating one sporulating parasite; the protoplasm of the cell is very much enlarged.

A similar account of this case, illustrated by a coloured plate, will be found in the *Lancet* of July 4th, 1908.

Cases of sporulating malignant parasites in the peripheral blood are rare. Cropper believes they must be commoner in some countries than in others. The case in question was from Palestine. As a rule, also, immature crescents are not seen in the peripheral circulation. Rosettes and sporulating forms in the white cells are usually seen in the large mononuclears, but Manson mentioned a case of a complete sporulating form in a polymorphonuclear leucocyte.

One may mention here the excellent German blood atlas of Meyer and Rieder[3] which contains good plates of the three types of malarial parasites and shows forms not usually seen in illustrations. These authors hold that it is possible to distinguish the young gametes in the red blood corpuscles. Special attention is directed to a ring form of gamete which only occurs in pernicious malaria. As regards benign tertian gametes, the young forms are recognised by the absence or relative insignificance of the nutrition vacuole, the older forms by their size, the compactness of their form (the protoplasm almost never showing amœboid prolongations) and their undivided nuclei. The occurrence of multi-nucleated gametes is rare.

Reference may here be made to Cropper's bodies, which are so apt to be mistaken in fresh films for malarial plasmodia. These were first described by Ross[4]* in India, then by

[1] Dr. Thiroux (September, 1906), "Des Relations de la Fièvre Tropicale avec la Quarte et la Tierce." *Annals de l'Institut Pasteur*, p. 766, Vol. XX.

[2] Cropper, J. (March 16th, 1908), "Phenomenal Abundance of Parasites in the Peripheral Circulation of a Fatal Case of Pernicious Malaria." *Journal of Tropical Medicine and Hygiene*, p. 91.

[3] Meyer, E., and Rieder, H. (1907). *Atlas der Klinischen Mikroskopie des Blutes.* Leipzig.

[4] Ross, R. (February, 1896). *Indian Medical Gazette.*

* Article not consulted in the original.

Malaria— *continued*

Cropper[1] in Palestine, and by Smith[2*] in the United States. A good account of these rod, spindle-shaped and motile melon-seed bodies is given in an article on Piroplasmosis by Nuttall and Graham-Smith,[3] who observed the bodies in the blood of normal dogs and of those suffering from the disease.

In Palestine they appear to be specially numerous in the blood of persons suffering from so-called "Syrian Fever," which is not malarial (*see* "Fevers," *page* 66). Smith also found these bodies associated with fever. They cannot be well stained. Indeed it is difficult to stain them at all, and so far their true nature and significance is undetermined.

It is quite possible that some of the febrile cases met with in Khartoum, and from which dried blood films are invariably sent to the laboratories for examination, might be found to exhibit these bodies if opportunities for examining fresh films were presented.

Secondly, one may consider papers dealing with malaria from a more or less general point of view, and lastly with those indicating prophylaxis and treatment.

James[4] has argued that malaria, as met with in India, is, in reality, a benign disease, and that the serious effects and fatal results attributed to it in the past should in large measure have been laid to the charge of kala-azar. He points out that in malaria the tendency is not towards cachexia and death, but to the acquiring of immunity; thus, "natives who have resided during a number of years in a malarious place acquire an immunity to malarial fever."

Doubtless in Africa, as well as in India, the cachexia of kala-azar has been in the past attributed to malaria, but none the less it is certain that pernicious African malaria does produce a chronic cachectic condition and may be a very fatal disease. At the same time, in the Sudan, deaths from acute malaria or chronic malarial cachexia are, I believe, not very common, and there is probably more than a grain of truth in the arguments adduced by Captain James.

At the same time, we find that Rogers[5] thinks that 20 to 25 per cent. of the total fever mortality in India is due to malaria, and he speaks definitely as to the terrible mortality from malaria amongst native children. He gives a very full description of the disease as met with in India, from which we cull the following information :—

1. The condition of the tongue aids the diagnosis, for the furring, whether marked or otherwise, is uniform in distribution, and does not show the red edges of enteric and seven days' fever tongue.

2. The pulse rate helps, for one rarely gets a slow rate accompanying a high temperature, which is very frequently the case in seven days' fever.

3. In true malarial cachexia the liver may be markedly enlarged even down to the navel.

4. In order to get the characteristic temperature curves, four hourly charts should be kept.

5. The longest period Rogers has seen a fever showing malarial parasites in the blood, under efficient quinine treatment, is six days, a point of great practical importance.

6. In India, Rogers believes there is no evidence to show that distinctive quotidian fevers exist.

7. The prolonged rise of temperature (24 to 36 hours) is characteristic of malignant tertian, and is the most distinguishing clinical point between it and the benign forms.

8. In benign tertian malaria in India a double infection is the general rule, and typical single tertian charts are quite exceptional.

9. Quartan malaria is much rarer than the other forms, and a double type is the most common variety of infection in India.

10. Malarial fevers in India are not more persistent under adequate quinine treatment than those of Europe and America, hence, provided the dose is adequate and is being assimilated, undue persistence of fever points to another or an additional cause.

11. As a rule there is a diminution in the number of the leucocytes, though sometimes even a leucocytosis may be encountered when an enormous number of parasites are present. The important point is the ratio of the white cells to the red in malarial cachexia. It rarely falls to lower than 1 white to 1000 red, while in kala-azar the ratio is almost always below 1 to 1500 and is frequently much lower than this figure.

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[1] Cropper, J. (May 1st, 1905), "Note on a Form of Malarial Parasite found in and around Jerusalem." *Journal of Tropical Medicine*, p. 132, Vol. VIII., and *Journal of the Royal Institute of Public Health*, February, 1907.

[2] Smith, A. M. (October 7th, 1905), *Am. Med.*, p. 607.

[3] Nuttall, G. H. F., and Graham-Smith, G. S. (October, 1906), "Canine Piroplasmosis." *Journal of Hygiene*, p. 586, Vol. VI.

[4] James, S. P. (1905), "On Kala-azar, Malaria and Malarial Cachexia." *Scientific Memoirs of the Government of India*, No. 19.

[5] Rogers, L., "Fevers in the Tropics."

* Article not consulted in the original.

12. When kala-azar and seven days' fever occur the large mononuclear increase is not much of a diagnostic aid, but it helps to exclude early enteric. The increase is less marked during high fever than when the temperature is normal and it is distinctly more marked and frequent in benign than in malignant tertians, because in the former the blood film is more often made when the temperature is normal than in the latter where the apyretic periods are very short. **Malaria—** *continue*

Plehn[1]* regards the supposed immunity of the black races as a mere tolerance, a symbiosis of the parasite and the organism, a relative immunity, the equilibrium of which is liable to be disturbed by change of climate, excessive exertion, etc. This particular resistance to the malarial virus seems to be special, in a sense, for the black races. It appears to result from a tolerance to the toxines, which will often commence in the fœtus for if the parasites themselves cannot traverse the placenta presumably the toxines can do so.

In this connection, however, one would cite Moffat's[2] case of undoubted congenital malaria, while the following quotation from Sir Thomas Browne, quoted by Monro,[3] is so quaint and withal so convincing that one perhaps may be pardoned for inserting it here. The date is 1679 :—

Mr. John Earle's wife, Sir Ralph Hare's sister, fell into a genuine quartan, when she was yong with child, which held her long, and when she came to her time she was delivered of a daughter in the fitt, and the child was so ill that none thought she would live; butt it grewe better; butt, on the third day about the hower of its birth, fell into a fitt of an ague, and so agayne the third day after, and that quartane settled and lasted for divers moneths till she was emaciated to skinne and bone, and was so lowe that she was fayne to use bathing for a good while; but, she is now of the age of 18 yeares and a very full young woeman. I called to mind this account, because shee dined with mee lately; her father was your loving friend.

Returning to Plehn and his views on immunity, we find that he thinks that Europeans can acquire a relative immunity analogous to that of negroes by preventive quinisation.

D'Allocco[4]* describes a case of malaria with cerebellar symptoms, *i.e.* speech slow, difficult and scanning in character, anxious expression, marked tremor, deliberation and inco-ordination on attempting voluntary movements, whether of upper or lower limbs. This intention tremor was more marked on attempting delicate movements. Worthy of note is the fact that when the patient got over the fear of standing alone she could stand for a long time without fatigue.

Brun[5]* observed in Constantinople the frequent presence of an area of total dullness of the apex of the lung in one or other side in patients who are infected by malaria. Although the condition is ushered in with marked febrile symptoms there are no râles or crepitations to be heard. Quinine rapidly dispels the consolidations as a rule, but in some cases the dull patch does not clear up, remaining in a passive state permanently. This is rather an important matter as, under such circumstances, it would be easy to make a mistaken diagnosis.

Burgess[6] discusses the question as to whether there is such a thing as malarial pneumonia, and from his Indian experience decides that there is. In this view he is supported by Paterson.[7] The latter speaks highly of the value of effervescing quinine in such conditions.

It is interesting to note that among the Nilotic negroes of the Southern Sudan, practically all of whom as children probably suffer from malaria (*vide* Dr. Wenyon's report—Third Report), pneumonia is a common and frequently fatal complaint. Whether or not there is any relationship between the two conditions I cannot say, but the matter might repay investigation by medical officers at southern stations.

Passing now to general measures, personal prophylaxis and treatment, one may note at the outset Sambon's advocacy of hyperparasitism as an ally of man in his struggle with the malarial parasite. The brown spores of Ross have been shown to be a species of *Nosema*, and to be parasitic upon the malarial parasite in the mosquito.

As regards general measures, various recent papers may be consulted, for example those

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[1] Plehn, A., *Archiv. f. Schiffs. u. Trop. Hyg.*, Vol X., No. 2.

[2] Moffat, R. N. (May 4th, 1907), "Congenital Malaria." *British Medical Journal*, p. 1054, Vol I.

[3] Monro, T. K. (June 8th, 1907), "Congenital Malaria." *British Medical Journal*, p. 1396, Vol. I.

[4] D'Allocco, O. (January 5th, 1907). *Rif. Med.*

[5] Brun, H. de, *Presse Med.*, Vol. XV., Nos. 32–34. Quoted in Epit., *British Medical Journal*, April 27th, 1907.

[6] Burgess, J. H. (April, 1907), "Malarial Pneumonia." *Indian Medical Gazette*, p. 131.

[7] Paterson, J. F. (February, 1908), "Malarial Pneumonia." *Indian Medical Gazette*, p. 75.

* Article not consulted in the original.

Malaria—*continued*

read[1] at the 1907 meeting of the British Medical Association. Simpson states that, as regards the flight of Anophelines, a distance of a quarter of a mile is a sufficiently protective zone. In Africa, the brothers Sergent[2]* found that the maximum flight of Anophelines is less than a mile. Simpson mentions the use of contour catchwater ditches for dealing with low-lying, water-logged land, such as marshes at the foot of hills.

Ziemann summarises the methods which can be adopted for extirpating the malaria parasites in man as follows:—

(1) *a*. By systematic prophylaxis with quinine of those infected with malaria; *b*. by killing those malaria parasites which invade the human body at the beginning of their development and before a fever attack has set in—that is real quinine prophylaxis—(2) by extirpating those mosquitoes which convey malaria; (3) by protecting man against the bites of malaria mosquitoes; (4) by endeavouring to raise the resisting power of malaria-infected people by social prophylaxis—by care for the better feeding, housing, clothing and instruction in the nature of malaria, etc.

These headings are considered in detail. Here we need only note that "it is obvious that for an effective quinine prophylaxis only serviceable, easily soluble quinine (for example, hydrochloride of quinine) is to be used, and that the intestines absorb the quinine. In this case it is indifferent whether the quinine be taken in wafers, tabloids or gelatin capsules." The Sergents[3]* point out that the taste of quinine can be entirely masked by suspending it in olive oil.

As regards the various methods of administration, Ziemann states with reference to them all that an absolute protection against malaria, that is, against the first infection, is not to be effected by quinine alone, *i.e.* without at the same time, injuring the body. As a result of large experience, he recommends the so-called four-day universal prophylaxis which permits of the necessary quinine individualisation.

He prescribes:—

(*a*) 1 gramme quinine every four days, best given in the evening before going to bed, with 5 drops of acid hydrochlor. in a glass of water, and on the appearance of ringing in the ears and nervous troubles, with one gramme of potassium bromide; (*b*) 1 gramme euchinin every four days, if one gramme quinine cannot be endured; (*c*) ½ gramme quinine if 1 gramme euchinin cannot be endured; (*d*) ½ gramme euchinin if ½ gramme quinine cannot be endured. Those who cannot endure ½ gramme euchinin had better not go to the Tropics at all. In order that the quinine days may not be forgotten, he recommends that the drug be taken on the 1st, 4th, 8th, 12th, etc., in short, on all dates divisible by 4 up to the 28th, and then begun again on the first of the following month. This method is applicable to all countries, whether they be the seat of milder or severe malaria and to all persons.

If, despite the quinine, malaria be acquired, he still gives, for three days after the successful recovery from fever, 1 gramme quinine daily, then every second day for the next fourteen days and again every fourth day.

In cases of unsuppressible vomiting and diarrhœa, he recommends most strongly the intramuscular injection of quinine bi-hydrochloride (1 gramme in sterilised solution in the proportion of 1 to 2 of water) into the glutei. A sure absorption of quinine thereby takes place. Excellent results from these procedures are recorded.

Rogers believes in a prophylactic dose of from 10 to 15 grains in an adult and at least 5 grains in a child over five years of age. It should be given twice a week, either on two successive days, or every third and fourth day alternately, in acid solution, and never in pill form.

For information regarding the methods of Morgenroth, Celli, Koch and Plehn, the reader is referred to notes in the *Medical Annual*, 1907, pp. 366–367.

Ziemann has not seen the smallest permanent success from the application of ointments and oils supposed to prevent mosquitoes from biting. Some help at the most for a few hours.

In the Sudan I have tried a mixture of citronella oil and alcohol and the preparation known as Anti-kito cream, which contains eucalyptus. Both do some good, but neither can protect for any length of time. One of the latest of these remedies is a composite oil, the use of which is strongly advocated in Ceylon,[4] especially for coolies. It consists of citronella, kerosine and cocoanut oils, with a certain proportion of carbolic acid. Vaseline can be substituted for the cocoanut oil. The mixture is a limpid liquid, smelling only of citronella and has a far more lasting effect than either kerosine or cocoanut oil by itself. There is no harmful or unpleasant action on the skin.

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[1] Simpson, W. J. R. (October 19th, 1907), "Anti-malarial Sanitation."

[2] Sergents, Ed. and Et. (November, 1905), *Compt. Rend. de la Soc. de Biol.*

[3] Sergents, Ed. and Et. (*loc. cit.*)

[4] "A Simple Preventative against Malaria." Sept. 15th, 1906. *Journal of Tropical Medicine*, p. 283.

* Article not consulted in the original.

Malaria— *continued*

A very useful general paper which, in many ways applies to the conditions prevalent in the Sudan, is that by Howard,[1] on "Malarial Prophylaxis in Small Communities in British Central Africa." Special attention may be directed to what is said regarding the selection of sites for posts and mission stations. Anyone who has seen the posts on the Upper White Nile must agree that, however convenient from the point of view of trade and general communication, they are as a rule badly placed from a sanitary standpoint. The problem is frequently one of much difficulty, but one has often wondered why Kodok remains a seat of Government, when both for it and for Taufikia the site occupied by Malakal would have been infinitely more preferable. Melut, however, is well placed. Of course one is here looking at the question solely from the outlook of a sanitarian, and there may be good and weighty reasons for retaining stations alongside khors, which at certain seasons teem with the larvæ of anophelines. The Shilluk villages are, as a rule, situated a considerable distance from the Nile, doubtless to secure freedom from the winged hosts which are apt to render life unendurable.

Darker,[2] of Southern Nigeria, has a paper on what he calls intracellular injections of quinine in malaria. In place of a mixture of quinine sulphate and vaseline, which he was wont to employ in the case of negro children, he now injects a warm dense solution of quinine hydrochloride (neutral salt).

Ten drops of water are placed in a test-tube and 15 grains of quinine hydrochloride crystals are added. Shake. Heat till solution is complete. Boil. Cover tube with sterile cover till the end containing the fluid is "just warm" to the hand. Inject in the usual way either into the cellular tissue under the skin of the anterior abdominal wall in the case of a child able to walk, or into the deep tissues of the outer side of the thigh in the case of a baby in arms. Close needle puncture with a saturated solution of gum mastiche in rectified spirit and apply a piece of lint. The quinine solidifies (so it is said) in the tissues, and is absorbed in about two months, during which time the child's general appearance greatly improves.

When a number of children are to be treated, the test-tubes containing the solution can be kept in readiness in a water bath at 100° F. The temperature of the fluid in the syringe must not fall much below 100° F., or solidification will occur and re-heating and washing out be required.

In a similar way euquinine dissolved in rectified spirit may be employed, but the asepsis is more difficult.

Darker also remarks that dense solutions of the acid salts of quinine (those used for intramuscular injection in cases of malignant malaria) disorganise the tissues when they are injected and so are not absorbed as quickly as may be desired. Hence he suggests that, in pernicious cases, the quinine should be in dilute solution, say 1 grain in 10 drops of water, several injections being made at the same time if necessary.

Several letters on the hypodermic use of quinine in malaria appeared recently in the *Indian Medical Gazette*. Some are very practical and useful, and may be quoted from with advantage. Thus, Scott[3] says:—

The form in which quinine is most usually given hypodermically or rather intramuscularly is, I suppose, the quininæ hydrochloridum acidum of the B.P. dissolved in distilled water to the strength of 1 in 1 or 1 in 2.

I use the former strength, *i.e.* equal parts of quinine and water. My usual dose of gr. x can then be contained in the full barrel of an ordinary 20-minim hypodermic syringe.

I have also used quinine sulphate extensively for injection and found it perfectly satisfactory. I have thought that it caused more after-pain than the acid hydrochloride, but could not be sure of this. I dissolve it with tartaric acid. By warming in a test-tube gr. x of quinine sulph. can be dissolved by gr. iii of tartaric acid in m. 20 of water. Grains 5 of tartaric acid should be used if the solution is to be kept at all, otherwise the quinine will be precipitated. The solution will not keep so long as that of the acid hydrochloride anyhow. Of the sulphate, I always use a 1 in 3 solution, and fill the barrel of the syringe twice without removing the needle from the gluteal muscles.

Several cases of tetanus have occurred from injection of quinine, and absolute asepsis is essential. A boon to tropical practitioners would be a small pocket case on the lines of B. W. & Co.'s urine-testing pocket-case, containing a spirit lamp, a small vessel for heating oil on a stand over the lamp and just large enough to take an intramuscular needle. Another small vessel with a handle and flat bottom for boiling the quinine solution, a hypodermic syringe (all metal), a pair of forceps for taking the needle out of the hot oil, and three bottles to

[1] Howard, R. (January 1st, 1908), "Malarial Prophylaxis in small Communities in British Central Africa." *Journal of Tropical Medicine and Hygiene*, p. 1, Vol. XI.

[2] Darker, G. F. (December 1st, 1906), "Intracellular Injections of Quinine in Malaria." *British Journal*, p. 1577. Vol. II.

[3] Scott, L. B. (March, 1907), "The Hypodermic Use of Quinine." *Indian*

Malaria— *continued*

contain quinine solution (or soloids), castor oil for heating, and solution for sterilising the skin. The syringe is sterilised by drawing the hot oil up into it three or four times. The oil is at the right temperature when a blade of grass fizzles the moment it touches its surface. I use an improvised case containing these things in a tobacco tin.

I consider that the intramuscular administration of quinine is of most use as a preventive of frequently recurring ague attacks, or to obviate relapse after the cure of an attack of malarial fever. I have known it to put a stop to recurrent ague attacks, where dosage by the mouth had failed. I have not found it of special advantage in curing malarial fever.

Moncrieff[1] remarks :—

My experience of the various quinine salts I used hypodermically was as follows:—

1. Hydrobromide.—Solubility 1 in 24 of water. Moderately irritating and not sufficiently soluble. Dissolved with difficulty in hot water and became precipitated again as the fluid cooled.

2. Acid hydrochloride.—Solubility 1 in 6 of water. I only used this salt once, as it caused much irritation that persisted for a long time. I also injected it into my own arm and found it painful and irritating.

3. Hydrochloro-carbamide or urea-quinine.—Very soluble and unirritating.

4. Acid hydrobromide.—Solubility 1 in 6 of water. I found this the least irritating of the salts I used. I made an injection into my own thigh, and next day could scarcely find the place without looking for the puncture mark.

This quinine salt is quoted in few of the catalogues of drugs that I have seen. I found it described in "The Extra Pharmacopœia," Tenth Edition. Treacher & Co. obtained it for me with some difficulty.

If a solution be made of strength 1 grain quin. hydrobrom. acid to 7½ minims of water, the excess of water allows the thorough sterilisation by boiling without the risk of the salt becoming precipitated. Contrary to what one would expect, quinine is more easily given to an infant hypodermically than by the mouth; at least, such was my experience when using the acid hydrobromide or urea-quinine. For infants the needle should be sharp and of small calibre and the syringe should have finger-grips. I found the fine Schimmel needles very convenient.

Till recently, I was under the impression that hypodermic injection is, next to intravenous injection, the most rapid way of getting quinine into the circulation, but recent observations show that this is at least improbable. With hypodermic injection, as compared with internal administration, the longer the continuance of the fever, the comparative absence of cinchonism, the slow excretion of quinine and the not infrequent persistence of irritation at the seat of inoculation, all these favour the view that quinine is absorbed slowly when given hypodermically.

Further knowledge about this important matter is much needed; for if this recent view be correct, the giving of quinine hypodermically will only be indicated when its exhibition by the mouth is contra-indicated or in cases of severity when the two methods might be combined, as suggested by Captain Megaw.

Holt writes that relatively much larger doses of quinine are required for children than for adults, and that an infant of a year will usually require from 8 to 12 grains of the sulphate or 10 to 14 grains of the bisulphate daily. He occasionally gives double this quantity.

Williamson[2] states that he finds "A. & H. 'Aseptic slab,' 1*s.* 6*d.*, invaluable for any such hypodermic administrations, as the depressions are easily sterilised by the flame and all measuring saved as they accurately hold 5 and 60 minims respectively, and hold the syringe upright while the skin is being purified, etc."

Symons[3] declares that he can fully endorse the efficiency of administering the drug "with the needle."

The salt used is the acid hydrochloride of quinine which will dissolve in equal parts of *distilled water*. This solution is made up in the dispensary of the hospital, in an ounce bottle, and used when required.

The technique is as follows:—

1. A small hypodermic syringe is used, the needle of which is sterilised by boiling for 2–3 minutes in a test tube. The syringe is washed out with 1 in 20 carbolic lotion by means of drawing up some of the lotion into the syringe 3–4 times. A small spoon is also placed in the 1 in 20 carbolic lotion and is used to receive the quinine lotion when it is poured out from the bottle previous to changing the syringe. The glass stopper, together with the neck and mouth of the bottle, are thoroughly cleaned with a sponge dipped in 1 in 20, and the part, into which the solution is to be injected is, of course, prepared in the usual way. I consider all the above details absolutely essential, especially the cleansing of the bottle—a point likely to be forgotten.

The dose is 10 minims, equal to 10 grains of the salt, intramuscularly in the deltoid muscle. If it be given hypodermically, trouble in the shape of a superficial abscess may arise; never, however, when introduced into the muscle. As to tetanus, such a disease should never deter one from intramuscular injections, if the above precautions be taken. I have been injected in the deltoid on many occasions about 10 A.M., and have played polo the same evening, which speaks for itself as regards after local effects.

Sometimes a slight aching sensation occurs, whilst the solution is being injected, but it passes off immediately.

To my mind there is no comparison in the two methods, *i.e.* by injection, and by the mouth. By the former method you make absolutely sure of the patient receiving the dose of quinine which you adminster, and you do not derange the digestive organs. 3rd, the patient must have quickly come under the influence of the drug—a very important factor in "malignant" cases. The temperature comes to normal in 24–30 hours and stays there. In my wards the usual practice is to inject on three successive days and then on alternate days for the week, to make sure of the patient being quinised.

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[1] Moncrieff, W. E. S. (March, 1907), "The Hypodermic Use of Quinine." *Indian Medical Gazette*, p. 114.

[2] Williamson, J. R. (March, 1907), "The Hypodermic Use of Quinine." *Indian Medical Gazette*, p. 115.

[3] Symons, T. H. (May, 1907), "The Hypodermic Use of Quinine." *Indian Medical Gazette*, p. 191.

I have never seen symptoms of cinchonism from this method. I would add that all the cases are diagnosed by the finding of the plasmodium malariæ before the quinine is given, even if it means the patient remaining a few days in the wards before he receives any specific treatment. **Malaria—** *continued*

Rogers gives data to show that 10 grain doses three times a day are sufficient to cut short an ordinary attack of malaria in one to four days, while four to six such doses in the course of the twenty-four hours do not have any more rapid effect, although they are advisable if the infection is found by the microscope to be a severe one. In children (he says, confirming Holt) there is a tendency to give too small doses of this drug. One grain for each year of age may safely be given two or three times a day up to the age of 10, so that over 10 years a full adult dose should be given twice a day. Infants may receive 2 or 3 grain doses twice a day.

Strychnine is valuable to counteract depression.

As a general rule the drug should be given without regard to the temperature and without waiting for an intermission of the fever, but it may sometimes be advisable to throw in a larger dose, such as 15 or 20 grains, during a remission or intermission of the pyrexia.

Rogers does not find that, when given hypodermically, the drug acts more effectively and rapidly. He utters a warning against the risk of tetanus, suggesting that quinine may act in symbiosis with the tetanus bacillus, or possibly by paralysing the phagocytes and thus favouring the bacillary action. He much prefers intravenous injections which should always be used when unusually severe infection is found by the microscope and before any cerebral symptoms have appeared. To wait for coma, it may be said, is usually to wait for death! The soluble bi-hydrochlorate should be given, preferably along with strychnine.

He mentions rectal injections administered high up the bowel, and I may mention that Dr. Daniels told me it was the rule in the Malay States to employ this method in all very severe cases with brain symptoms.

The drug should be continued in 20 to 30 grain daily doses for a week or two after the pyrexia ceases. Thereafter 10 grains a day for one month from the date of attack. Then prophylactic doses twice a week as already indicated, for two months, or until the end of the malarial season if still exposed to infection.

Euquinine is indicated when gastric or intestinal catarrh is present, for the treatment of children or where quinine causes ill effects; 15 grains is the dose for adults.

One finds little said about quinine given in effervescing form, but personally I have found that quinine and citric acid, given along with carbonate of ammonia and potassium bicarbonate in an effervescing mixture, is not only very efficacious in malaria but after the attack acts as an excellent tonic, improving appetite and imparting energy. I am inclined to agree with Burney Yeo that one gets the full effect of the drug with smaller doses when it is administered in this fashion, and that it is more easily retained and assimilated.

The acetyl-salicylate of quinine has recently appeared and is said to be useful, the effect of the drug being obtained only when the salt reaches the alkaline contents of the intestine.

Carpenter[1]* strongly advocates fresh splenic extract given as powder in capsules in 5 grain doses every four hours. In quartan and estivo-autumnal types a hæmatinic is usually required in addition, but in the acute tertian and quotidian forms the splenic extract alone is sufficient.

Slatincano and Galesesco[2]* report favourably on atoxyl injection (doses 50 cgr.) in cases of tertian infection. Apparently the single injection was followed by complete cure.

For enlarged malarial spleens Johnston[3] finds that injections of bisulphate of quinine with iron tonics internally, and the application of flying blisters, is much to be preferred to the old routine treatment of iron and quinine internally and the local application of red iodide ointment.

Since the above was written, a very important paper by Celli,[4] on the campaign against malaria in Italy, has appeared. As regards prophylaxis, he differs in certain respects from

[1] Carpenter, C. R. (August 4th, 1906). *Medical Record.*

[2] Slatincano, A., and Galesesco, P. (December 14th, 1907). *Compt. R. Soc. Biol.*

[3] Johnston, C. A. (May, 1906), "Enlarged Spleen and Its Treatment." *Indian Medical Gazette*, p. 179.

[4] Celli, A. (April 1st, 1908), "The Campaign against Malaria in Italy." Translated by J. J. Eyre, *Journal of Tropical Medicine and Hygiene*, p. 101.

* Article not consulted in the original.

Malaria— *continued*

Ziemann and others who have been quoted. He also refers to several interesting facts not previously stated:—

1. The *Anopheles* are never wanting where the fevers exist, but their quantity is not always in direct proportion to the intensity of the epidemy; in fact it is frequently in inverse proportion. On the other hand, there may be microbes and *Anopheles* without malaria developing itself, even when malarial patients arrive there from other places, or some autochthonous or sporadic case of fever manifests itself there. Microbes and *Anopheles* may therefore persist, and, notwithstanding this, the malaria may become attenuated and disappear.

2. The number of *Anopheles* infected is always small, even in the places and months most affected by the fevers. The hereditary transmission of the infection from mosquito to mosquito has not been demonstrated up to now.

3. There is no doubt that quinine acts in inverse proportion to the degree of development of the malarial parasites in the blood stream; that is to say, it acts best against the sporozoites directly they are inoculated and least against the sexual forms destined to maintain the recurrent fevers, and little or not at all against the sexual forms destined to propagate the species. Thus some fevers are pertinacious in recurring in spite of the abundant and protracted use of quinine, either alone or associated with iron and arsenic. In fact, these latter drugs under whatever form and in whatever way administered have no value as direct anti-malarial remedies.

4. Quinine, provided it be *administered daily*, is in average and even therapeutic doses better tolerated, and for a longer time than, *a priori*, one could have believed; that is to say, after the first two or three days it no longer produces the least singing in the ears, and is not only completely innocuous but also acts as an aid to nutrition and as a tonic to the digestive apparatus and muscles, thereby increasing the appetite and the power of work. Quinine taken daily is always present in the blood, and thus prevents instead of produces the phenomena of quinism. Further, there is not, perhaps, another example of a remedy so perfect, nor one which so rapidly establishes itself, and can be prolonged for a long time (up to five or six months), and yet can be interrupted when desirable without any disturbance, and without, although the organism is habituated to the small and average doses, diminishing the curative efficacy of the large doses when they are necessary.

5. Intolerance is rare if a salt insoluble in water such as the tannate of quinine be given. It is specially indicated for young children, and it is important to give it in the form of comfits or chocolates.

6. Dosage—40 centigrams of the bisulphate, hydrochlorate or bi-hydrochlorate for adults and young persons; 20 centigrams of the same salts or 30 of the tannate of quinine for children. In districts with very severe malaria, 50 to 60 centigrams of the bisulphate may be given.

It will be seen that this method differs very considerably from the others quoted. Certainly the statistics given by Celli speak well for its efficacy, but it must not be forgotten that a procedure suitable for Italy may not be equally so for tropical Africa. At the same time, I do not know that this Italian system of quinine administration has ever been given a fair trial in the Dark Continent.

Malta Fever. Most of the recent important work on this subject is embodied in the Reports of the Special Commission which studied Malta Fever in all its aspects at Malta.

A useful *résumé* is given by Bruce.[1] He mentions briefly the long course (average four months), the extreme irregularity of the temperature curve, the exacerbations of the fever, the presence of symptoms of a rheumatic or neuralgic character, and the extreme anæmia and debility characterising the tedious return to health.

He notes the disappearance of the disease from Gibraltar, and its occurrence in Malta, Tunis, Alexandria, South Africa, Rhodesia (where it apparently followed the introduction of goats), India, China, the Philippine Islands and America. He might also have mentioned the Anglo-Egyptian Sudan, for it has been proved beyond all doubt to occur in this country, chiefly, so far as I know, in the Kassala Province and the northern districts. I have been able to confirm the diagnosis in at least one case by the agglutination test. The other special points to which allusion is made may be tabulated:—

1. Broadly speaking, the better the social position, the greater the liability to the disease.

2. In Malta, the disease is prevalent all the year round, being commonest during the coldest and rainiest months. It is markedly regular in its appearance, a large number suddenly cropping up in February, December, or other of the cold and rainy months.

3. Its distribution is general as regards the population of Malta.

4. The principal path by which the *Micrococcus melitensis* leaves the body is the urinary tract. The urine sometimes contains the organisms in enormous numbers. It may also quit the body in the blood to a small extent by the agency of mosquitoes and other blood-sucking insects.

5. The micrococcus is fairly resistant to external influences. It can exist in a dry condition in dust or clothing for two to three months. It lives in tap-water or sea-water for about one month. It can live a week even in urine which has decomposed and become markedly alkaline. Exposure to sunlight kills it in a few hours.

6. No habitat outside the body, such as sewer air, dust, harbour water and other insanitary media could be found.

7. Infection by contact or by fomites was proved not to occur.

[1] Bruce, D. (March, 1907). *Journal of the Royal Army Medical Corps.*

8. Infection by contaminated dust was found rarely, if ever, to occur.

9. It was definitely proved that the organism in the vast majority of cases, reaches the human body by way of the alimentary canal.

10. Infection by the agency of mosquitoes and other biting-flies is of the rarest occurrence.

11. The infection, as is now well known, takes place by the ingestion of contaminated goat's milk. Ten per cent. of the Maltese goats were found to secrete the micrococci in their milk and 50 per cent. of these animals gave the specific agglutination test when their bloods were examined.

Malta fever disappeared from Gibraltar because the Maltese goat has disappeared from that station.

12. Another mode of infection is probably by the absorption of the urine of infected patients.

13. Preventive measures, on the lines indicated by this important discovery, have already resulted in very marked benefit, and there is every hope that the disease will be stamped out from the garrison in Malta.

Basset-Smith,[1] in a somewhat similar and more recent review, mentions in addition :—

(*a*) The constant presence of the organism in the peripheral blood of cases suffering from the disease.

(*b*) The presence of the infective organism in the urine of apparently healthy men.

(*c*) The infection of other domestic animals (mules and dogs), although goats are those chiefly implicated.

(*d*) The occurrence of localised epidemics.

(*e*) Complete cessation of cases where infected milk was removed from the dietary or when it was properly sterilised.

(*f*) The question of "protective inoculation."—This still remains *sub judice*.

(*g*) The question of treatment. The use of anti-sera and vaccines has proved disappointing. As regards drugs, quinine in large doses is harmful. Salicylate of quinine may do good in combating the insomnia and persistent neuritis of the later stages. Arsenic and iron are indicated for the cachexial and strychnine for cardiac irritation.

(*h*) Pathologically there is evidence that the condition is a general septicæmia, as witness various local lesions such as ulceration of the small intestine.

Leishman[2] considers that it is very desirable that investigation should be directed towards ascertaining the line of communication from diseased to healthy goats. He suggests that the disease might be attacked by immunising those animals by means of injections of attenuated cultures of the micrococcus, and cites the good results achieved by this method in cholera (Haffkine) and in plague (Strong).

Davies[3] doubts if milk is the only source of infection, and has seen cases which suggest that the mosquito may be a vector. He found that the disease was four times as common in children who consumed unboiled milk as in those drinking boiled milk.

Williams[4]* cites a case of a woman, suffering from Malta fever, who gave birth to an infected child. The colostrum contained *M. melitensis*. On the fifth day after birth, the child's blood was found to give the agglutination reaction in a dilution of 1 in 500.

Brayne[5] gives a good account of 26 cases met with in India. They departed from the classic type met with in the Mediterranean in many particulars. The following are the chief points noted, and one records them because it is very probable that cases in the Sudan will be found to conform to this Indian type, the climatic conditions and general surroundings in both countries being more or less similar. Brayne's cases all gave the agglutination test :—

1. The fever is irregular, lasting any time from one week to two months.

2. There was a tendency in the severer cases for the fever to be of a high-continued or remittent type for two weeks or so, and, if the patient lives, to then assume an intermittent character.

3. There was a great tendency towards the rapid development of anæmia.

4. The pulse was noted to bear no relation to temperature, thus a weak dicrotic pulse of 108 was often seen with a practically normal temperature, and within half-an-hour the rate might have fallen to 60.

5. The very profuse sweating was most characteristic, and as a result the patients had a characteristic smell.

6. There was complete absence of joint symptoms.

7. The spleen was somewhat characteristic, in the majority of cases, being just palpable, very hard and, if tender, very slightly so.

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[1] Basset-Smith, P. W. (January 4th, 1908), "Recent Work on the Cause, Prevention and Treatment of Mediterranean Fever." *Lancet*, p. 21.

[2] Leishman, W. B. (January 4th, 1908), "Recent Work on the Cause, Prevention and Treatment of Mediterranean Fever." *Lancet*, p. 21.

[3] Davies, A. M. (January 4th, 1908), "Recent Work on the Cause, Prevention and Treatment of Mediterranean Fever." *Lancet*, p. 21.

[4] Williams, E. M. (July, 1904), "Mediterranean Fever: Infection in Utero." *Journal of the Royal Army Medical Corps*, Vol. IX., No. 1.

[5] Brayne, F. W. (December, 1907), "Notes on 26 Cases of Malta Fever in the Native of India." *Indian Medical Gazette*, p. 441.

* Article not consulted in the original.

Malta Fever *—continued*

Rogers[1] deals with Malta Fever in India but practically, owing to lack of material, gives an account of the disease as seen in Europe. He has, however, a note on the degree of dilution desirable for the agglutination test in India. He prefers to put up the blood in dilutions of 1 in 40, 1 in 80 and 1 in 160, and to look only on 1 in 80 as a certainly diagnostic reaction, and 1 in 40 as a suspicious one necessitating re-testing at a later date in higher dilutions. The reaction appears within a few days of the commencement of the fever, and appears to persist fairly constantly throughout its course, and for some time after convalescence is established.

Kennedy[2] has a paper on the bacteriology and etiology of the disease. He gives the chief naked eye appearances of a two to five days' culture of *M. melitensis* as—its transparency and amber colour by transmitted light, its white globular appearance by reflected light, and a beautiful phosphorescent green shadow which is cast through the medium by direct light, and is best seen by looking sideways through the medium. The micrococcus is killed in ten minutes by dry heat at 90° C. to 95° C.; by moist heat at 57·5° C.; by 1–2000 hydrarg. perchlor. and by 1–100 phenol; in one hour by sunlight, 130° F. (54·4° C.); in four to five hours by 1·2 per cent. Clayton gas or 0·7 per cent. liquid SO_2. An important practical point mentioned is that it may be recovered from the three weeks' old urine of a patient, or from the clothes on which the urine has dried.

On artificial media it retains its vitality for a very long time, having been recovered from an agar culture 820 days old. In recovering it from a litmus milk culture during a period of nine months (284 days), Kennedy noted that about the fifth month it lost its character of emulsifying and remained clumped like a staphylococcus, but about the eighth month it recovered its normal characteristics. He also states that in doing the agglutination test it was not at all infrequent to find that 1–100 gave a complete reaction immediately, 1–50 took longer, and 1–10 and 1–20 gave no reaction or only a trace (paradoxical agglutination reaction).

Zammit[3] found that the agglutination test could be applied to the milk of infected goats as well as to the blood. The technique is as follows :—A strong emulsion of the *M. melitensis* is prepared in normal saline solution in a watch-glass. To this a small quantity of formaldehyde solution is added (one small loopful of a 1 per cent. solution), the whole being drawn into a pipette. The formaldehyde prevents the milk turning sour. One drop of the emulsion is placed on a glass slide and a loopful of milk is mixed thoroughly into it. This mixture is then drawn up into a fine capillary pipette, left in an upright position for 12 hours, and the reaction noted at the end of that time. The reaction is often seen after a few minutes. The cream collects at the surface and does not interfere with the reaction.

Critten[4] draws attention to the difficulty in diagnosis from early pulmonary tubercle, for while tubercular disease does not induce the formation of substances capable of agglutinating *M. melitensis* in 1–10 and 1–20 dilutions, still, owing to co-existent or past infection with *M. melitensis*, the serum of a patient suffering from tubercle of the lung may clump the micrococcus and the possibility of tubercle should always be excluded by careful clinical and bacteriological examinations.

The organism has been obtained from small quantities of blood, hence vein puncture may not be necessary, for, if the blood be collected in the usual way, with proper precautions in a large, curved, collecting tube into which a little five per cent. citrate of soda has previously been introduced, and the blood be then expelled into a broth flask and incubated at 37° C., growth may result.

Birt[5] has some useful notes on the agglutination test. He says :—

It is essential to make use of a recently-isolated culture or one grown on a medium which does not induce auto-agglutinability or sensibility to the agglutinins of normal blood. I have found that emulsions of growths on glucose nutrose agar of +25 reaction (Eyre's scale), though isolated more than a year, are still satisfactory. When emulsions of old laboratory cultures on ordinary agar are made with physiological salt solution, no clumping may be apparent, yet a minute trace of human serum from any source may agglutinate the micrococci completely. Hence it is incumbent on the bacteriologist to control his emulsion by testing it with normal human blood. A reliable culture is usually unaffected by, and is never completely clumped by, a tenfold dilution of blood serum

[1] Rogers, L., "Fevers in the Tropics." 1908.

[2] Kennedy, J. C. (December, 1907), "Remarks on the Bacteriology and Etiology of Malta Fever." *Journal of the Royal Institute of Public Health*, p. 728, Vol. 15, No. 12.

[3] Zammit, T. (February, 1908). *Comm. Rep.*, Part IV. p. 98.

[4] Critten, A. (June 1st, 1907), "Some Observations on Blood Serum Reaction in Tubercle and Mediterranean Malta." *Journal of Tropical Medicine and Hygiene*, p. 187, Vol. X.

[5] C. (November 9th, 1907), "Mediterranean Fever in South Africa." *British Medical Journal*, p. 1336.

derived from healthy people or from those suffering from any disease except Malta fever. Nor are traces of a reaction observed in a twenty-fold dilution. Therefore agglutination of the *Micrococcus melitensis* by a serum diluted twenty times, is diagnostic of Mediterranean fever, past or present. Of all serum tests, it is most reliable, since we are not embarrassed by "coagglutinins" which may complicate the diagnosis of typhoid and para-typhoid fevers. Thus, during the course of a typhoid infection, the body may elaborate coagglutinins for the para-typhoid bacillus and vice-versa. In my experience, emulsions killed by heating to 55° C. for one hour, with 0·5 per cent. phenol added afterwards, are more generally useful than suspensions of the living micrococci. With the former I have not observed the occurrence of "agglutinoids," which I have occasionally encountered when the latter have been employed. That is to say, while the lowest dilution of the serum failed to clump the living micrococci, the higher dilutions agglutinated them, notwithstanding that the serum was in less amount.

Eyre[1] reviews the whole question of Melitensis Septicæmia in the Milroy Lectures for this year. He describes acute, subacute and ambulatory types and enters carefully into a consideration of its effects on the different systems, giving an analysis of clinical symptoms met with in 1000 cases. The morbid anatomy is detailed, special mention being made of the numerous globuliferous cells found in sections of the spleen, liver and kidney. They are derived from the endothelium lining blood sinuses, and contain in their interior from one to 15 or 20 red blood discs. The increase of lymphoid tissue in the spleen is noted and the bone marrow is stated to be of a typical lympho-erythroblastic type (presence of giant cells, mononuclears, lymphocytes with dimunition of myelocytes and polymorphs) as opposed to the leucoblastic marrow associated with pneumococcic infections.

The technique for the agglutination reaction both macroscopic and microscopic is described. As regards the latter, Eyre himself requires a positive reaction in a dilution of 1 in 30 to 1 in 50, preferably the latter, within half an hour. He cites certain precautions which must be observed in carrying out the test:—

1. The serum should be clear and free from blood discs. 2. The culture of *Micrococcus melitensis* employed should be one recently isolated from the human body (or recently passed through a laboratory animal) and grown on agar of + 8 or + 10 reaction and incubated at 37° C. for not longer than two or three days—a 24 hours' old culture giving the most reliable results.

He notes that—

Old cultures, or cultures many generations removed from the animal body, are prone to agglutinate automatically in the presence of normal serum, or normal saline solution, or even when simply suspended in distilled water.

The so-called "pro-agglutinoid" zones are more common in *Micrococcus melitensis* than, for example, in enteric, *i.e.* a serum will yield a good reaction when tested against *Micrococcus melitensis* in, for example, dilutions of 1 in 30, 1 in 40, 1 in 60, 1 in 80, but will fail completely to clump the cocci in dilutions of 1 in 50, or will react in dilutions from 1 in 50 upwards to 1 in 500, and fail to react in lower dilutions such as 1 in 10 and 1 in 20. It is therefore essential to prepare and observe a *series* of dilutions in performing the test for diagnostic purposes.

He gives the technique for the recovery of the organism from the spleen, the blood, the urine and the fæces. As regards the blood examination, he thinks the blood should be collected from a vein of the arm *late in the day*, when the patient's temperature tends to be high and at or near the height of a pyrexial attack.

Withdraw 5 c.c. into a sterile syringe already containing a few drops of 10 per cent. sodium citrate solution. Transfer the citrated blood to a test-tube or small flask containing 45 c.c. of nutrient broth, and incubate at 37° C. From the third to the tenth day of incubation an agar slope-tube must be inseminated from the broth culture, and itself incubated for from three to seven days before a negative result can be recorded.

The work of the Commission is reviewed and evidence advanced to prove that the micrococcus can be transmitted by sexual congress. Eyre considers that the mosquito may certainly act as a carrier, and suggests an unusual mode of possible infection through a skin abrasion from the infected excrement of mosquitoes, a method similar to that described by Lamb in the case of the flea and plague (*vide* "Plague," *page* 162). There are very interesting notes regarding the goat and its history, and with reference to the mechanism of goat infection from the milking methods in vogue. Lack of space forbids a more minute review of this section, but one may note that the progeny of infected milch goats is itself uninfected at birth. This is a point in connection with the preventive measures advocated. A modification of the "Bang process" for the reproduction of tuberculous herds is recommended for Maltese goats, and Eyre believes if this were carried out under scientific supervision the whole of the infective animals seized could probably be replaced in three or four years by healthy goats. The flesh of infected goats is harmless when cooked. Enough has been said to show that these lectures contain much of interest and value and will well repay careful perusal.

Recent Italian work, dealing with the vitality of the *M. melitensis* in various beverages, the histo-pathology of the disease, etc., will be found mentioned in the *Journal of Tropical Medicine and Hygiene* for July 15th, 1908.

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[1] Eyre, J. W. H. (June 13th, 20th and 27th, 1908), "Melitensis Septicæmia." *Lancet*, Vol. I.

Measles. Khartoum has once suffered from an epidemic of measles, and that the disease is one of great importance in tropical countries is evident from the report[1]* of the Sanitary Commissioner for the United Provinces of India in 1903, wherein it is stated that in 23 districts, measles was returned as one of the chief causes of infantile mortality. In all it accounted for no less than 98,112 deaths, being present in true epidemic form.

The point which chiefly concerns us is the nature of the more modern methods taken for its prevention, but in the first place attention may be drawn to the work of Hektoen[2]* on experimental measles, which led him to conclude that the virus of measles is present in the blood of patients with typical measles some time at least during the first 30 hours of the eruption; furthermore, that the virus retains its virulence for at least 24 hours, when such blood is inoculated into ascites broth and kept at 37° C. At the same time, the exact nature of the virus has not been determined, and Hektoen's work is open to criticism as no mention is made of Koplik's spots occurring in the persons inoculated with blood from measles patients, although they seem to have developed typical catarrhal symptoms.

Rosenberger[3]* blistered the skin during the eruptive stage in measles, and in the fresh serum thus obtained found in 39 out of 41 cases.

A more or less constant hyaline body, possessing the following characteristics. In size it varied from 1/10 to 1/6 of the diameter of a red blood-cell. It was generally spheroid, but ovoid or pyriform forms were also seen. It was, as stated before, perfectly hyaline, and possessed, or had attached to it, a round or irregular oval-shaped granule of a brownish-black colour, which was actively motile. This granule travelled round the entire periphery of the body in a most deliberate manner, stopping every now and then, and appearing to try to gain an entrance into the body. In two or three instances flagella were seen, but never more than two. In the same specimen of fluid another hyaline body, also motile, but containing two to four small motile granules, was occasionally seen. Attempts to cultivate the body were made, but without success. Serum from normal persons, a few cases of scarlet fever, and a number of syphilitics, did not contain this body.

Koplik's spots have been mentioned. Their value in diagnosis has been shown by Bruening,[4]* who found them present in 50 cases of measles out of 52 examined. They were found from six days to one day before the appearance of the rash, and were not present in many cases of German measles, scarlet fever and serum eruptions.

The best account one has seen of Koplik's spots occurs in a review of Bing's[5] work, "*Les Taches de Koplik, leur Importance pour le diagnostic et la Prophylaxie de la Rougeole.*"

We quote it in full:—

In its typical and characteristic form, Koplik's sign consists of a macule and a papule; the macule is a rose spot of irregular, star-like, or rounded outline; the whitish-blue papule forms the central point of it and is alone the characteristic and pathognomonic constituent of the spot. It is usually only just large enough to be visible and never exceeds a diameter of one millimetre, consequently the statements often made that this central spot is from two to five or more millimetres in diameter, show that other buccal eruptions are frequently mistaken for it. This papule projects slightly and can be felt with the finger; it is associated with a slightly rough or lustreless appearance (*"aspect dépoli"*) of the mucous membrane, which is often of great value, since it appears quite early and persists after the actual spots have disappeared. The number of spots varies within very wide limits, being in some cases only one and in others several hundreds. After a time the rose spots tend to run together and coalesce, but the white centres maintain their individuality. They are found only upon the mucous membrane of the lips and cheeks, and not, as some few authors have described, upon the gums, the soft palate and the tongue. Dr. Bing maintains that such spots are the erythemato-pultaceous stomatitis described by Comby, and not Koplik's spots; moreover, small punctiform vesicles occur upon the palate in a variety of conditions, including rubeola, scarlet fever and influenza, as described by Koplik himself. The buccal eruption appears to go through a definite cycle which may take from two to six days to accomplish, but the disappearance is more or less abrupt since the small white spots become easily detachable and are removed by the buccal secretions, while ulceration never occurs. The diagnosis and the nature of the sign are carefully described and exhaustively discussed by Dr. Bing, who then details his own observations. He concludes that the sign is pathognomonic of measles, but that its absence does not prove that the patient does not suffer from that disease. With regard to the time of its appearance, he finds that it may appear from one to five days before the eruption, but most often two days before; it reaches its maximum on the day on which the rash first appears on the face, commences to disappear on the full efflorescence of the rash, and does so completely from the second to the fifth day of the eruptive stage. He further considers the value of the sign in diagnosis and, after an exhaustive study of the conditions likely to be mistaken for measles, arrives at the conclusions that it is a sign of the utmost value, and that in every case of illness in a child it should be sought, since it enables isolation to be carried out sooner than would be possible otherwise. The character of the spots has no bearing upon prognosis or on the severity of the attack.

As regards prevention, a recent and excellent article will be found in M'Vail's[6] work. He points out that the infection, which is very evanescent, is transmitted by the air and

[1] Quoted in *Indian Medical Gazette*, I. 1905. Vol. XL.

[2] Hektoen, L. (March, 1905), "Experimental Measles." *Journal of Infectious Diseases*, Vol. II.

[3] Rosenberger, R. C. (June, 1906), "American Medicine." Quoted in *Medical Annual* for 1907.

[4] Bruening, H. (1906). *Deut. Med. Woch.*, Bd. XXXI., No. 10.

[5] *Lancet* (May 18th, 1907). Vol. I.

[6] M'Vail, J. C. (1907), "The Prevention of Infectious Diseases."

* Article not consulted in the original.

that is not easy to establish the supposed rôle of the cat as a carrier. He asks what are the causes of the failure to stamp out measles, and replies :— **Measles—** *continued*

In the first place, though its infectivity is evanescent, yet while it lasts, measles is one of the most infectious of all diseases. It is a very rare thing to see a single case of measles. Nearly every first case produces a crop of others. In the second place, it is very infectious before the eruption appears, and therefore before the disease is recognised as measles. When a medical man is called in, the presence on the buccal mucous membrane of Koplik's spots, which often appear a day or two before the rash, may aid diagnosis, but the difficulty is that very many cases are not seen by a doctor at this stage. Thirdly, parents look on measles as of no consequence, and often do not send for a doctor. Fourthly, very few local authorities apply to the disease the powers of the Compulsory Notification Act. Fifthly, epidemics come on so quickly and so extensively that hardly any authority has a staff sufficient to cope with them. Sixthly, as compared with small-pox, there is no vaccine to protect individuals against attack. Seventhly, school attendance gives the infection every facility for spreading, and countries where education is compulsory are correspondingly liable to measles.

To the question, Is hospital isolation the remedy? the reply is in the negative owing to the nature of the disease and the prohibitive cost. Notification is useless if no action follows it. It is noteworthy, however, that broadly speaking, the disease is decidedly dangerous up till the beginning of the fifth year of life; is very little dangerous from that time to the end of the tenth year, and afterwards is free from danger to life. The policy, therefore, is to delay attack by measles—and measles is spread mainly by schools. What is all-important is to receive early information of its occurrence. A couple of useful pages on the vexed question of school closure are here inserted in full as likely to be of value, for in Khartoum there are both Government and mission schools, and much might be done by prompt action to avert or mitigate a measles epidemic :—

With reference to the circumstances under which there is reason to hope that closure of a school, or of the infant department of a school, will be effective in arresting the spread of measles, the general dictum may be taken to be closure on account of the first case present in school during the infective stage. If it should so happen that a first case is detained at home by parents from the onset of catarrhal symptoms, or if a first case develop catarrhal symptoms on a weekly holiday and does not return to school thereafter, the child may be regarded as not having been in school during the infective phase, and a medical officer may correctly stay his hand from closure. But if a first case has been present, say, in the infant department, red-eyed and sneezing, that first case is an indication for closure of the department.

In determining the time for which a school, or part of a school, should be closed, with the object of checking measles, the medical officer will again be guided by circumstances; but his ultimate criterion will be the reputed incubation limit of measles, which may, for practical purposes, be taken as ten to fourteen days. It follows that the beginning of closure cannot be delayed without risk much longer than a week from the date of the last presence in the school of an infective case, for the case may have been infective for two days or three before it ceased attendance. It follows also that the end of closure need not be carried beyond fifteen days from the same date, which allows a margin of safety. It may occur in actual practice, more frequently than not, and especially in the absence of notification, that a case of measles escapes the notice of the medical officer of health during its earlier days. Alertness on the part of school officials may effect much towards obviating this omission, but in most instances a few days will have passed before the data for closure are before the medical officer.

Under these conditions, the medical officer will observe, in the first place, that, despite the lapse of a few days, he may still have a day or two in hand before closure need take effect. Incubating measles is not infectious, and these few days of grace will give him time to make the necessary arrangements, or to explain to incredulous officials the benefits which will follow the step proposed. It is not essential that he should justify the scientific faith that is in him by postponing closure to the limit of safety, but the knowledge that closure need not in all cases instantly follow the discovery of an infective child may prove helpful to him at a time of stress.

The medical officer will observe, in the second place, as suggested above, that is is not necessary to continue closure beyond fifteen days from the last exposure of the infant department to infection. An example will make these general statements plain. On 3rd June, the medical officer becomes aware that the child is at home with measles. Now the earliest date at which a case is probable will be ten days from the first exposure of the school children on 29th May, that is to say, 8th June, and the latest date at which a case is probable will be fourteen days from the last exposure of the school on 31st May, that is to say, 14th June. The medical officer decides on closure. He closes the department on the 5th or 6th of June, with two or three safe days in hand, and he keeps it closed from the 5th or 6th to the 15th June, a period of nine or ten days in all.

At present it is not very often that the disease is heard of early enough to make this system of dealing with it practicable. And, even where notification is compulsory, parents frequently do not send for a doctor at all, so that medical notification is a broken reed, while notification by parents is almost entirely neglected. But much more is likely to be done in the future than has been attempted hitherto for prevention of spread of measles by schools.

M'Vail remarks, however, that the introduction of regular medical inspection of school children is the likeliest way to prevent the spread of measles by means of schools, and that great assistance is to be expected from an intelligent and painstaking teacher, as the latter has an excellent opportunity of observing the case in catarrhal stage. Naturally school closure is of much less value in towns than in the country, for in the latter case there are less opportunities for close intercourse out of school hours. Valuable notes on school ventilation complete an able paper, which has attached to it an appendix in the form of a type of an informative pamphlet, which we reproduce, as it is excellent.

Precautions against the Spread of Measles

A dangerous disease.—Measles is popularly looked upon as a trifling disease. That is a very serious mistake. Nowadays far more children die from measles than from scarlet fever. It is especially dangerous to very young children, and the longer the disease can be warded off in childhood the greater are the chances of recovery.

Its early intimation.—Its notification is not compulsory, but in the interests of infected families, and of the public generally, it is very important that the earliest possible intimation of its existence should be made through the sanitary inspectors.

Its symptoms.—To make early intimation practicable, early recognition of the disease is necessary. It begins with the signs of a bad cold in the head, a hard cough, running at the nose, sneezing, and tender and watery eyes. The rash appears first on the face and spreads from it to other parts of the body. It consists of red blotches, and the face looks swollen. It seldom appears until three or four days after the first catarrhal symptoms, yet the disease is very infectious from the beginning. Measles is indeed amongst the most infectious of all diseases.

Isolation of cases.—Wherever, therefore, its invasion appears likely, parents and teachers should be on the look-out for the first manifestations of measles, and whenever a child is attacked by what appears to be a cold in the head, it should at once be isolated and all precautions taken against spread of infection. Mild and severe cases are equally infectious. A medical man should be sent for in every case, because it is often a fatal blunder to regard the disease as not dangerous. The only safe place for the child is in bed, where it should be kept warm, as most of the deaths are due to chest complications developing from the supposed "cold." At the same time, as noted below, the room should be reasonably ventilated. Isolation should be maintained for at least a fortnight from the appearance of the rash, and the child should not return to school for at least another week. Complications may readily extend these periods.

The sick room.—The patient should have a room to himself, and whoever is in attendance on him should avoid mixing with the rest of the household. Unnecessary articles of furniture should be removed from the sick room, and a fire should be kept burning both to warm the air and to promote ventilation. The windows should be kept a little open at the top, but the bed should be out of reach of a draught.

The patient should have his own cups, plates, glasses, spoons, knives, forks, etc., which should be kept in the room and washed there.

Disinfection.—All bed and body linen, handkerchiefs, pinafores, and other such articles should be disinfected before being removed from the sick room. They may be disinfected either by steeping in a solution provided for the purpose, or by boiling in water. Discharges from the throat and nose should be received in old handkerchiefs or pieces of linen or cotton, which should afterwards be disinfected as above or burned if worthless. Any person necessarily visiting the sick room should cover his clothing with a loose coat and should avoid contact with the patient and the bedclothes, and should wash his hands before leaving the room.

Cleansing.—After the patient has recovered, the whole house and its contents should be thoroughly cleaned—floor, walls, woodwork, furniture, bed and body clothing. Soap and water should be abundantly used. The house should also be well aired by opening the windows.

A paper on the same lines as M'Vail's, but bringing out some additional points, will be found in the *British Medical Journal*, 27th January, 1906, where the conclusions of a careful enquiry into the whole question are given. These are that :—

1. Measles at present in London only spreads in classes under five years of age, except in certain better class districts. Seventy-five per cent. of children above five in infants' departments are protected.

2. Measles tends to spread whenever a class accumulates unprotected members to the extent of between 30 per cent. and 40 per cent., and when spread has begun it continues until the proportion is reduced to between 15 per cent. to 20 per cent. unprotected.

3. If children under five were excluded from school, closure for measles would not be necessary for some time in London, except in one or two special districts; if, however, this were successful in postponing attacks, the question would again become acute in a few years.

4. To deal efficiently with measles, accurate knowledge is required; the measles history should be elicited for each child on admission to school. This should be done universally; if notification to the school authority was made compulsory on every parent whose children attended school, our hands would be greatly strengthened. Unless the amount of susceptible material is fairly accurately known for each school, we shall only be working in the dark, as heretofore, and nothing of any value can be expected to accrue from our methods.

5. To effect any useful purpose, school closure must take place before the "first crop" falls. The old practice of waiting until the attendance fell to a certain limit was useless in arresting the spread of measles, and did absolutely no good.

6. The means of arresting spread of measles, other than school closure, are of enormous importance. To name two—sanitary buildings and training of teachers; these two factors probably have the greatest effect of all in determining the extent of spread of an outbreak.

7. Deaths often occur from ignorance on the parts of parents. Circulars should be sent out wherever measles has appeared in a class, that is, on the incidence of the first case, begging mothers to notice colds, and upon the slightest suspicion of such symptoms to keep the child at home for a day or two. In this way, measles would probably be checked to a far greater extent than has been effected by any other means. Instances have come to our notice where heartbroken mothers have stated that, had they known, they would have treated colds seriously, of which they took no note, and thereby their children's lives would have been saved.

8. Measles never spreads in the boys' and girls' departments. There is no need to exclude children from infected homes who attend the upper departments.

Measles— *continued*

A more recent delivery on the subject is that of Buchan,[1] who thinks that the more recent method of excluding only the infants and susceptible older children seems that which is most gaining ground. In epidemic times, daily medical inspection of those children attending the infant departments is indicated, but too much must not be expected from this measure, and in the future the school teacher, properly trained, is likely to be of the greatest service. Lastly, the question of disinfection after measles falls to be considered. M'Vail contents himself with thorough washing and cleansing, scrubbing of floors and furniture, scouring of bed and body clothing, and the free admission of fresh air.

Brown[2] sums up the advantages and disadvantages of disinfection as a prophylactic measure in measles in the following terms. The advantages appear to be :—

1. The prevention of the spread of the disease by infected articles in a very small and problematical percentage of cases.
2. Its educational effect, *i.e.* inculcating on parents the serious nature of measles.

Among the disadvantages may be mentioned :—

1. The very small part it plays in preventing the spread of the disease.
2. The small return for the labour and expense of carrying it out.
3. The piecemeal method in which it can be done at the best.
4. The annoyance which this latter causes.
5. The difficulty of diagnosis; and lastly,
6. Its leading to concealment of cases.

As regards its educational advantage, it seems to me a very expensive and ineffective method of education, and this can be much better done by our method of forwarding in every case precautions to be taken in the disease, as well as an extract from the Public Health Acts bearing on the prevention of infectious diseases, followed by a visit from the Sanitary Inspector.

On the whole, the disadvantages appear to me to outweigh the advantages, and probably the best method would be to do it on request by the medical man in attendance and in those special cases where circumstances point to its utility.

In a later paper,[3] he recommends :—

That general disinfection after measles be discontinued, and that it only be done on the request of medical men in attendance, the parents and guardians, or in special cases where circumstances point to its utility.

Milk. One has reference to a very large number of papers on milk and milk supply, but most of those considered here will be found to have a bearing on the question as it affects tropical countries.

Blackham[4] quotes Giles, who, in his book on "Climate and Health in Hot Countries," says :—

Goats are extremely hardy and, being naturally clean feeders, require far less attention than cows, while the flavour of their milk in tea is preferred by many to that of cows' milk. They stand marching well, too, and are therefore better suited for use in camp; and as their favourite food is the leaves of bushes, they may be trusted to find their living to a great extent as they trot along on their way from camp to camp. Usually their milk agrees excellently with infants, but there can be little doubt that asses' milk is superior for this purpose.

Goats' milk requires somewhat less dilution than that of the cow, and may agree in cases where cows' milk fails.

Asses' milk is probably the best substitute for an infant's natural food, and, failing this, the goats' milk is to be preferred to that from the cow.

Blackham himself confirms Giles' opinion that the milk of a "clean feeder" is much more desirable than that of an animal which occasionally finds its nutriment in village rubbish heaps. He says :—

If Indian mothers realised that when pressed by hunger there is no fouler feeder than a cow, and it is dismal fact that in the polity of an Indian village, the cattle rival the pigs in their efficiency as scavengers, they would, when travelling about and unable to keep a cow of their own, prefer the milk of the goat to that of the cow as food for their infants.

Most of the Khartoum milk supply is derived from goats and in itself is of good quality and flavour, but it is only fair to state that I have seen herds of goats browsing on rubbish heaps containing mostly straw and paper, while the conditions of supply leave much to be desired and will not be improved until the scheme indicated under "Sanitary Notes" (Third Report) can be carried into effect.

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[1] Buchan, J. J. (April, 1908), "Preventive Measures in Measles." *Journal of the Royal Institute of Public Health*, Vol. XVI.

[2] Brown, R. K. (January, 1907), "Disinfection after Measles as a Preventive Measure against its Spread." *Public Health*, Vol. XIX.

[3] Brown, R. K. (January, 1908), (*loc. cit.*)

[4] Blackham, R. J. (September 29th, 1906), "Goats' Milk for Infants." *Lancet*, Vol. II.

Milk— *continued*

Cahill[1] points out that goats' milk is superior to cows' milk as a food for infants. It is primarily more digestible because its casein forms only a flocculent curd, and the infant does not suffer from the accumulation of hard cheesy masses, as with cows' milk. The goat is singularly resistant to tuberculosis, and the nourishing power of its milk is just as light as that of cow's milk. He mentions that goats' milk has no unpleasant or peculiar smell or taste, provided the goat be kept under cleanly conditions and apart from any association with the male of its species. It is said to be a fact that when a he-goat is allowed to run with a herd the females acquire, for the time, something of the characteristic smell of the male and that their milk becomes similarly affected. Wright[2] also praises goats' milk as a food for infants. He contrasts the goat, a cleanly animal, with the byre-stalled cow, the reverse. The fæces of the former are practically solid and rolled in balls so as to prevent any possibility of their adhering to its hind quarters. On this account these parts, as also the udder, are always fresh and clean. Goats dislike filthy surroundings and will not lie down amongst their excreta nor eat soiled fodder.

The goat is practically immune to tuberculosis, and, with but few exceptions, the only instance where they have been found to be affected are when they have been closely housed with tuberculous cows, from which they have derived their infection. This being so, and the animal being small and hardy, goats can be kept in pens within the city without detriment to their health, although it might be advisable to let them loose in the open once a year, where they can live on any uncultivated land and clean it for cultivation in a very short time. Wright also gives some very interesting quotations and statistics, and cites Place, who, after much experience, says :—

> Anyone who will take the trouble to look up the data will readily see that in those countries where the goat is domesticated and its milk is used in the family, there is very little tuberculosis, almost no scrofulous glands, and the infant mortality naturally is decidedly less for those children who use the milk.

It is also said that the statement regarding the alleged odour of goats' milk is erroneous. If the goat be allowed to roam about and to eat weeds, twigs and all kinds of vegetation at will the milk is apt to be very strong in odour. On the contrary, however, if the animal be fed purposely for obtaining palatable milk, no odour can possibly be detected.

Hook says : "The milk from goats fed on an English meadow on the roadside has no flavour to distinguish it from cows' milk."

As it is probable that the supply of milk obtained chiefly from cows at the Government farm will increase in the future, some notes from a useful and practical paper by Kinsella[3] may be quoted with advantage. Dealing with the care and aeration of milk, he first considers :—

> *Flavours in Milk.*—Ordinarily speaking, we have two classes of injurious flavours in milk to contend with. Those are food and contamination. All those flavours of various foods which are fed to the cow, and which the milk absorbs from the animal before being milked, are termed "food flavours." As a rule such flavours are more pronounced at the time of milking.
>
> Contamination flavours are those which gain access to the milk after it leaves the udder of the cow. These latter flavours are of two kinds, or rather come from two sources : one is due to the flavours of certain substances which are absorbed by the milk after milking ; the other is due to the milk being directly influenced by bacteria, which also takes places some time after the milking has been completed.
>
> *Food Flavours.*—Speaking from a practical point of view, food flavours cannot always be entirely eliminated, yet they can be minimised by judicious feeding and by proper aeration in a pure atmosphere. Food flavours are primarily due to the presence of volatile oils contained in the strong flavoured foods, and such flavours leave the animal through the different secretions of the body.
>
> When the feeding is done immediately after the milking, these food flavours largely pass off through the other secretions and are not so noticeable. On the contrary, when the feeding is done during the milking process, or shortly before, the larger portions of these objectionable flavours are thrown off from the body of the animal by means of the milk.
>
> When it is absolutely necessary to use such feeds as turnips and large quantities of maize ensilage, or other feed which causes such disagreeable flavours, the dairyman can lessen the trouble considerably by feeding with discretion as above recommended. If, also, some rough forage be fed along with the foods, which cause the objectionable flavours, it will assist in doing away with such flavours.
>
> *Contamination of Flavours.*—While feed flavours are to a certain extent beyond the control of the supplier or milk vendor, contamination flavours are entirely within his control ; but the great trouble is that suppliers to factories and city and town milk vendors frequently attribute such flavours to the feed the cows eat, in order to screen the lack of cleanliness.

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[1] Cahill, J. (October 6th, 1906), "Goats' Milk for Infants." *Lancet*, Vol. II.

[2] Wright, W. (November 3rd, 1906), "Infantile Mortality and Goats' Milk." *Lancet*, Vol. II.

[3] Kinsella, J. A. (1903), "City and Town Milk Supply, and the Care and Aeration of Milk." Transvaal Department of Agriculture, Bull, No. 6.

Milk—*continued*

The principal troubles with flavours, both for city and town supply and for the manufacture of butter and cheese known as contamination, are caused by bacterial infection. Those flavours due to the milk absorbing the flavours of certain strong substances to which it may be exposed are, as a rule, not nearly so objectionable as those caused by the action of the living germ. In fact, the predominant part of our flavours are due to the action of bacteria which gain access to the milk through lack of cleanliness in connection with the milking utensils, surroundings, methods of delivery, etc.

He also deals with :—

Milking—and how it should be done.—Milch cows should always be kept clean instead of being allowed, in fact as they often are compelled, to tramp through slush, mud and liquid manure.

Too frequently we find cows milked when in a dirty and unsuitable condition. Bacteriologists have frequently traced serious trouble, in connection with milk supplies, to causes produced by these and similar conditions. This dirt and manure often dries on the cow's udders, and readily finds its way into the milk-pail in the form of dust.

Great care should be taken to remove all dry particles of manure and filth from any place on the animal where it is likely to be dislodged into the milk-pail.

The udder and teats of the cow should be thoroughly cleaned with a damp cloth or washed before the milking is commenced.

The milking should be done with dry hands only, and the filthy practice of lubricating the hands with milk should under no circumstances be tolerated.

The hands of a milker should be washed before the milking is commenced. In no case should milkers with long filthy finger nails be allowed to milk cows.

In many cases the teats of cows are in this way poisoned, and the practice of pressing the ends of the fingers against the sides of the animal's teats is also objectionable.

During and after the milking, the most scrupulous cleanliness is necessary in order to keep the milk in the best condition. To attain this object the milk should be removed, immediately after milking, to some place where it would not be exposed to dust and bad flavours.

In connection with the handling of milk on the farm for butter and cheese factory purposes and where no suitable well-ventilated milk-house can be provided, it is always a wise precaution, in selecting a place for the milk-stand, to take into consideration from which direction the prevailing wind comes. The stand or place where the milk is kept should always be placed on the windward side of the cow-stable; besides this, it should be some distance away, in order that when the wind changes, or on close, still, and muggy nights, the milk may not become contaminated or deteriorated in flavour. The milk should be kept at least fifty yards from the stable or shed.

Straining of the Milk.—When the milking is completed, the milk should be immediately removed from the cow-stable and carefully strained through a proper wire, or very fine hair, strainer. A few doubles of clean butter cloth make an excellent strainer, but the objection to its use is that it is too frequently not properly washed and scalded.

A milk or cream strainer of any sort requires great attention, and unless they are thoroughly washed and scalded each day they become a source of contamination rather than a purifier.

Many dairymen and milk vendors are inclined to believe that the straining process is a cure for all dirt and filth that gets into the milk, and for that reason are less careful in the milking process than is necessary. They evidently forget that the bulk of the solid matter quickly dissolves on entering the milk-pail, and that the milk thereby becomes seeded with germs, which easily pass through any strainer and continue their work of deteriorating the flavour of the milk.

Aeration and Cooling.—In aerating milk on the farm for either city and town consumption, or for delivery to butter or cheese factories, it is essential that the aeration takes place in a pure atmosphere. Unless this be done the process may become a source of contamination.

The benefits of aeration, when carried out in a pure atmosphere, are two-fold: firstly, it facilitates the escape of certain food flavours; and, secondly, the cooling which takes place during the aeration retards the growth of germs which may have already gained access to the milk. If, as above stated, aeration cannot be carried out in a clean place where the air is fairly pure, I would recommend cooling the milk only.

Why we cool the milk is to check the growth of bacteria. If the milk be allowed to stand at a high temperature it produces conditions eminently favourable for the growth of those germs which produce bad flavours in all dairy produce.

The most desirable temperatures for the cooling of milk for either city consumption or for the manufacture of butter is from 50 degrees to 55 degrees; and although it is not always practicable to do this on the farm, every endeavour should be made to lower the temperature to as near this as possible.

Many species of bacteria which produce bad flavours in milk do not grow well at a temperature of 50 degrees, while those that do develop grow very slowly, and the development of those organisms which produce souring in milk are almost entirely stopped.

In hot climates, aeration on the farm will be found insufficient for the proper cooling of milk. When this is the case, it should be supplemented with cooling by passing the milk over a cooler through which cold water is running.

The cooling may also be done, though not so quickly or advantageously, by placing the cans or pails in a stream or dam where the water is fairly cold. If this be not available, the cans or pails can be placed in tanks of cold water and the temperature soon reduced by keeping the milk stirred with a dipper and by changing the water."

The paper is well illustrated, different kinds of sterilising ovens, milk cans and pails, etc., being figured.

Milk— *continued*

A recent work by Kenelm Winslow, worthy of attention, is "The Production and Handling of Clean Milk," in which the use of paper bottles is suggested as a means whereby much of the present trouble and expense in bottling milk would be obviated.

A point likely to be of considerable importance in the Tropics is brought out by Römer and Much,[1]* who discuss a non-bacterial cause for the deterioration of milk. They found :—

That milk which had been sterilised by the combined hydrogen peroxide and perhydrase treatment regularly acquired an unpleasant odour and taste if allowed to stand in the sunlight. Samples kept in the dark did not undergo this change, even after a considerable length of time. Similar results were obtained with raw milk, with milk that had been sterilised by steam, and with the powder of milk dried *in vacuo*. The cause for this peculiar change could therefore be neither a bacterium nor an enzyme. Further experiments showed that the change only occurred if sunlight and oxygen together were allowed to act on the milk. An idea as to the rapidity with which the deterioration sets in may be obtained from the following figures:—A flask of milk exposed to direct sunlight was spoiled after twenty minutes; another flask exposed to diffused daylight, only after five days; a third sample kept in the dark was good after a fortnight. The blue, violet and ultra-violet rays proved to be most active in inducing the change. This fact may become important in view of the recent suggestion to sterilise milk by the use of ultra-violet light.

With regard to the chemical nature of the phenomenon, it appears that only the cream undergoes the change, whilst solutions of the milk-casein and whey do not show it. Now, it was proved by Ritsert, in 1890, that the "rancidity" of butter-fat is due not so much to a bacterial action as to the combined effect of light and air. More detailed work by Jensen proved that the bacteria are mainly responsible for the hydrolytic change (acid production, "true rancidity"), but that sunlight and air have a marked oxidising effect, which is chemically evident by a decrease of the iodine number ("tallowy change").

The authors confirmed these results in their experiments, and point out the danger incurred, especially by infants' milk which is usually bottled and which may be often seen exposed to the sun.

This doubtless accounts for the peculiar taste which milk sometimes acquires in Khartoum, and which, I think, has sometimes been attributed to the goat when faulty storage was the true cause.

Mettam[2] deals with "Diseases of the Udder and the Milk Supply," pointing out that the mammary gland may be attacked by tuberculosis, actinomycosis, and botriomycosis, as well as by the more general organisms of suppuration. There is also a form of mastitis due to a streptococcus which is contagious, and though, according to Mettam, this streptococcus is not pathogenic for man, still the idea of drinking milk containing many streptococci is repellent.

He notes, however, that in acute mastitis the changes in the secretion and in the secretion gland are so marked that no sane owner of dairy stock would think for a moment of adding what he may obtain from the gland to the general output. As regards cow-pox, he thinks it would be dangerous to permit the distribution of the milk, because of the possibility of its becoming contaminated by lesions in the sinuses of the udder and the risk of children of tender age becoming infected by drinking the milk in a raw condition. He mentions the remarkable case of brown scabs upon the teats and udders of a cow, due in all probability to infection with organisms of human diphtheria, for a bacillus exactly resembling the Klebs-Lœffler organism was isolated from the lesions. Probably the local infection was of human origin.

There is a good deal said about tuberculosis which really comes under the heading "Veterinary Diseases" *(page 217)*, but mention may be made of the four methods employed for arriving at an opinion as to the condition of the udder. These are :—

(1) The application of tuberculin; (2) manual examination of the udder and its glands; (3) examination of the secretion by (*a*) microscopic examination after sedimentation and approximate staining, (*b*) animal experimentation; (4) harpooning the quarter and withdrawing a portion of the affected gland for examination.

(1) Application of the tuberculin test will only give a general reaction, and the knowledge gained from the results following its use will only be of value if the clinical evidence (2) is beyond doubt. It is not possible to ascertain during life unequivocal evidence of any local reaction.

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[1] Römer and Much (1906), *Berl. Klin. Woch.*, Nos. 30 and 31, quoted in *Journal of Preventive Medicine*, October, 1906, Vol. XIV.

[2] Mettam, A. E. (January, 1907), "Diseases of the Udder and the Milk Supply." *Journal of the Royal Institute of Public Health*, Vol. XV.

* Article not consulted in the original.

Milk— *continued*

(2) Physical examination of the udder and of its glands will greatly assist in arriving at a correct conclusion, if the reaction to tuberculin is positive.

(3) The microscopic examination of the deposits from milk after centrifugalising is not very satisfactory, especially as acid-fast bacteria indistinguishable from the tubercle bacilli may be present. Injection of the deposit into guinea-pigs with the object of inducing lesions is unsatisfactory, because of the length of time that must elapse before the lesions become manifest. For this reason I have not included the use of laboratory media for the isolation of the organisms, as the time occupied is too long and the results are too uncertain. If, however, the suspected organisms are relatively abundant, the isolation of the acid-fast bacteria may be attempted to ascertain if these develop within a few days; if they do, then they are not tubercle bacilli; if there be no development the negative evidence may be taken as favouring the diagnosis of tubercle.

(4) Harpooning the udder. Provided the owner of the animal is willing, there is no difficulty in carrying out this simple operation. It goes without saying that an aseptic operation is desirable. The tissue removed may be examined histologically for the tubercle bacilli and the characteristics of tuberculous lesions. The presence of acid-fast bacilli in the tissues, even if the anatomical elements are not clearly indicative of tuberculosis, is, in my opinion, sufficient for a positive diagnosis, inasmuch as the acid-fast bacilli of milk probably gain access to the milk after it has left the cow.

It must be noted, however, that Much[1]* has recently found in cattle forms of the tubercle bacillus which are not acid-fast, distinguishing two types—a rod-shaped form which is partly granular and a granular form consisting of granules lying singly or clustered into irregular groups. These were found in the lungs of infected cattle, and nothing is said about their presence in milk, but it is evident that such aberrant forms must now be taken into consideration.

Mammitis, due to the presence of streptococci, is discussed as is the finding of streptococci derived from abscess in the gland or merely from the surface of the udder.

They are not necessarily abundant unless the milk contains pus, and if it does, diagnosis is not difficult, because the pus cells are readily found in the precipitate following centrifugalisation. It would be well, in this connection, to remember that numerous cells, leucocytes and epithelial cells may be present free in the perfectly normal acini of the gland, and that these cells may be swept away by the secretion and appear in the milk. There is not much difficulty, however, in recognising the pus cells as distinguished from the leucocytes, and the number is always an admirable guide. Numerous cells will indicate pus or a catarrhal condition, few cells have no pathological significance.

This leads one to consider several papers on the significance of leucocytes and streptococci in milk.

Savage[2] believes that at—

The present day (1906) we are not in a position to frame satisfactory bacteriological standards for milk, and until more precise knowledge is acquired, it will not be possible for milk examination to take a place at all comparable to that which the bacteriological examination of water occupies. The significance of streptococci in milk, he says, is of great practical importance, and the presence of pus in milk is also of considerable importance. That milk should not contain pus few will deny, but, he asks, what constitutes pus in milk? All milk contains leucocytes. When does a leucocyte become a pus cell, and what distinguishes the one from the other? What number of leucocytes, or pus cells, constitutes pus in milk?

He sets himself to answer these questions.

The technique of the examination for streptococci and *B. coli*, and the method of enumerating leucocytes, is given. The result showed a striking prevalence of streptococci. In sixty-eight milk samples examined they were present in forty-five, or 66 per cent. when 1 c.c. of the milk was examined. They can only come from the interior of the udder or from unclean manipulation. This can be determined by the *B. coli* examination. The precise value and significance of these streptococci is difficult to determine, but it is noticeable that they more closely approximate to the streptococci most common in human fæces (Houston) than to those more frequently found in saliva (Gordon). In particular they agree with the former, in that they all, with one exception, were found to ferment salicin, while the great majority of streptococci from saliva, isolated by Gordon, fail to do so. Savage notes that if future work confirms this provisional deduction that all streptococci from cows' milk ferment salicin, then the finding of streptococci with this character in the throats of persons suffering from milk-carried streptococcal outbreaks may become valuable evidence, as showing that these streptococci were of bovine origin and causally connected with the outbreak. The work indicated that there was an absence of any relationship between leucocytes and streptococci, while Savage states that he was unable to differentiate between a leucocyte and a pus cell or to lay down an arbitrary standard as to what number of leucocytes per cubic millimetre is to be designated pus in the milk.

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[1] Much, H. (April 6th, 1908). *Berl. Klin. Woch.*

[2] Savage, W. G. (April, 1906), "Streptococci and Leucocytes in Milk." *Journal of Hygiene*, Vol. VI.

* Article not consulted in the original.

Milk— *continued*

As regards the presence of *B. coli*, Savage considers this organism as always derived from outside the udder and a definite indicator of contamination during milk collection or storage.

Harris[1] has reviewed the whole subject, quoting numerous authorities and concluding that :—

(*a*) The statements of Kruse, Hölling, and Heinemann cast considerable doubt upon the value heretofore entertained regarding the significance of streptococci in milk.

(*b*) It is not excluded by the evidence that pathogenic streptococci are to be found at times in milk; in fact, recorded observations make this certain, the contamination arising from clinically recognisable cases of mastitis in the herds.

(*c*) We are not as yet in possession of any reliable method for distinguishing a non-pathogenic from a pathogenic streptococcus.

(*d*) The sanitary significance of the so-called " pus cell " has been greatly overrated. More scientific attention should be given to the study of the phenomena of lactic leucocytosis, together with a more accurate method of enumeration, such as that of Doane and Buckley or of Savage.

(*e*) Particularly, should more attention be given to veterinary inspection of the cows' udders, with less absolute dependence upon laboratory examination of milk, for signs of infectious processes.

A still more recent paper is that by Pennington and Roberts,[2] who, dealing with Savage's statement as to leucocyte and pus cell, state :—

However, it does seem possible, in a large percentage of cases, to determine, by considering the relative number of the various kinds of leucocytes going to make up the cell content of the milk, whether they are polymorphonuclear, large or small mononuclear, eosinophiles, etc.; and by their staining and appearance, whether they are degenerated or in good condition morphologically, loose lying cells or collected into masses with evidences of fibrin; and from such observations it seems possible to determine whether the condition which produces them is likely to be pathological or normal; and finally summarise the results of their enquiries in Philadelphia as follows :—

A correlation of the finding of streptococci or of many leucocytes in milk by the laboratory, with the physical condition of the cows in a herd maintained for the production of a very clean milk, would seem to show that in many cases there is a connection between such findings and the condition of the cow, both in relation to specific udder and to systemic affections. Such parallelism seems to obtain for the end of the lactation period, for the beginning of an udder inflammation, for an attack of cow-pox, and, possibly, for chemotactic conditions due to high feeding of animals constitutionally unable to transform the increased feed into increased milk.

The frequent laboratory examination of the milk of individual cows has materially assisted the herdsman in preserving the good health of the animals and has stimulated more frequent and careful clinical observations.

It is believed, too, that such examinations are a very material factor in maintaining a bacterial count, which, for the past thirteen months, averages 3267 organisms per c.c. when the milk is from twenty-six to forty hours old.

An interesting paper is that by Robertson[3] on the milk supply of Edinburgh. He gives Houston's standard for specific organisms in milk.

(1) 1 c.c. should not give evidence of *Bacillus enteritidis sporogenes.*

(2) 0·001 c.c. should not give evidence of *Bacillus coli communis.*

(3) 0·001 c.c. should not give evidence of streptococci.

Primary sediment should not exceed, after twenty-four hours, 100 parts per 1,000,000 c.c.

Secondary sediment (after centrifugalisation) should not exceed 50 parts per 1,000,000 c.c.

In obtaining a pure milk we should strive to limit the number of micro-organisms to 20,000 per c.c., and if the milk is strained and refrigerated immediately after it is drawn and the fore-milk thrown away, there should not be great difficulty in attaining this perfection. He further states :—

If there are a great number of micro-organisms in the milk, it proves that the temperature of the milk has been kept too high, which is a condition favourable to the rapid development of germs in such a good breeding medium; and further, that the milk has been produced under insanitary and uncleanly conditions.

The special micro-organisms, *B. coli communis* and *B. enteritidis sporogenes*, afford direct evidence of the contamination of the milk with cow-dung or human excrement, and cause diarrhœa, and are therefore of special importance in the consideration of infantile mortality.

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[1] Harris, N. McL. (May, 1904), " The Relative Importance of Streptococci and Leucocytes in Milk." *Journal of Infectious Diseases.* Suppl. 3.

[2] Pennington, M. E., and Roberts, E. L. (January 30th, 1908), " The Significance of Leucocytes and Streptococci in the Production of a High Grade Milk." *Journal of Infectious Diseases*, Vol. V.

[3] Robertson, R. (October, 1907), " The Milk Supply of Edinburgh, with Suggestions for the Improvement of Milk Supplies Generally." *Journal of the Royal Institute of Public Health*, Vol. XV.

Milk—*continued*

The *B. enteritidis sporogenes* is of particular importance, as it does not multiply in the milk, and can therefore be accepted as an indication of the original pollution of the milk.

Streptococci should not be present, and show disease of teats or inflammatory conditions of milk glands. They are instrumental in causing sore throats, and give a standard of sanitary requirement for the country and for the town.

Revis[1] has also a paper on the detection of added water to milk, and after citing the three ways in which the defendant in a case may prove the contrary, none of which are very satisfactory, he states that :—

From a careful study of the records of analyses of genuine abnormal milks, of which a great number will be found scattered throughout the literature of milk, two general hypotheses may be formed, viz. :—

1. When genuine milk is deficient in non-fatty solids, the deficiency is due entirely to an abnormally low percentage of milk sugar, the proteids and ash being present in their normal amounts.

2. When a genuine milk shows an unusally high percentage of non-fatty solids, the increase is due almost entirely to an abnormally high percentage of the proteids, the sugar and ash either remaining normal or perhaps slightly increasing also.

With the latter hypothesis we are not here concerned. The former, however, is of immense importance, as it allows of a simple means of differentiating, among milks of low non-fatty solids, between a deficiency due to natural causes and causes distinctly fraudulent, such as the addition of water. Stated in a concise form, we may put it thus :—

If in the case of a milk in which the percentage of non-fatty solids is below 8·5 per cent., an estimation of the sugar gives a figure considerably less than 13/24 of the estimated non-fatty solids, and such that the total sum of non-fatty solids could only be present if the sugar were present with a normal amount of proteids and ash, we have very strong evidence that the deficiency of milk solid is due to natural causes only. If, on the other hand, the sugar figure approximates closely to 13/24 of the estimated non-fatty solids, the evidence is just as strong that the deficiency is due to fraudulent adulteration with water. The simple addition, therefore, of a sugar determination to the usual estimation of fat and total solids, furnishes the analyst with evidence of the most useful kind in deciding on the cause of a deficiency of non-fatty solids in a sample of milk.

The polarimetric estimation of sugar in milk is simple, rapid and exact, and there can be no possible objection to it. The only objection of any weight to the whole procedure that can be brought, is the difficulty of putting samples rapidly enough in the analyst's hands before lactic fermentation has destroyed an appreciable amount of sugar. Such a difficulty could be easily surmounted by the exercise of a little administrative capacity.

An article by Musgrave and Richmond[2] on infant feeding and its influence upon infant mortality in the Philippine Islands deals amongst other things with milk; human milk, goat's milk, cow's milk, caraboa's milk and various kinds of sterilised and preserved milks being considered. The paper has chiefly a local interest, but as some of the brands of milk examined have evidently a world-wide distribution and are to be found in the Sudan, reference to their analyses may be useful, while the rules for infant feeding in the Tropics are to be commended.

Passing now to methods for preserving milk, the most recent, and probably most valuable, method is that of Buddeization—an account of which appears in the *Lancet*, of December 14th, 1907. Put very briefly, it consists in cooling, which, however, must not be carried too far, because in excess it destroys the natural anti-bacterial qualities of the milk, heating to 50°C., centrifuging, again heating in a vat and adding peroxide of hydrogen. This acts both in virtue of its own strong germicidal power, and still more owing to the fact that the milk "catalase," an enzyme of the living cell, is able to decompose the hydrogen peroxide, setting free nascent oxygen, which has a still greater bactericidal action. 50° C. is found to be optimum temperature for this action. There is a stirrer in the vat, and when the sterilisation is complete the milk is either cooled first and then bottled, or run into sterile bottles and then cooled. As the presence of the catalase is not absolutely uniform, it is best to add so much peroxide of hydrogen that there is certain to be a trace left at the end of the process. Then immediately before bottling a few drops of catalase solution are added.

Milk, after having been subjected to the foregoing treatment, possessed the following qualities. None of the component parts of raw milk were in any way altered. The milk was practically sterile, and most specially so in regard to the specific pathogenic micro-organisms. The milk was able to be kept considerably longer than ordinary milk, but notwithstanding this fact it should, of course, like all milk, be consumed as fresh as possible. The milk was absolutely free from foreign substances. As for the milk enzymes, the tryptic and peptic proteases and the lipases were unimpaired. The oxydases were

[1] Revis, C. (January, 1907), "The Detection of Added Water in Milk." *Journal of the Royal Institute of Public Health*, Vol. XV.

[2] Musgrave, W. E., and Richmond, H. T. (August, 1907), "Infant Feeding and its Influence upon Infant Mortality in the Philippine Islands." *Philippine Journal of Science*, Vol. II., B.

Milk— *continued*

destroyed, but, seeing that these do not occur in human milk, this was generally considered of no importance. The catalase was destroyed. On the other hand, oxydases nearly always accompany catalase, so that by the final addition of a little catalase both these classes of enzymes were restored to the milk. The nutritive qualities were unaltered, and numerous cases had been recorded by medical men in which people (both infants and grown-up people) had been doing very badly on raw or cooked (Pasteurised and sterilised) milk, whereas milk thus treated had agreed very well with them. The taste and flavour of the milk were practically unaltered. Non-sporing organisms and the vegetative forms of sporogene bacilli are killed by the process. Regarding the spores there is some difference of opinion, but in its latest improved form it would seem that spores can be killed with certainty. It would seem also that the process renders the milk more digestible.

There are many other valuable papers, but the above are the most recent in the list, and, possibly, the most useful to the reader in the Tropics. One may conclude by noting an account[1] of the "Victoria" brand of homogeneous fresh milk, said to be of special value in the Tropics. It is a fluid, not a condensed milk, and is guaranteed free from preservatives, chemicals, sugar, or any added matter whatsoever. It is a pure milk without additions, and in such a state that it can be used at once as it is poured from the tin. The slight flavour induced by it in the process of preparation is said to be pleasant and by some is regarded as an improvement. I have seen and tasted a sample of this milk after it had been one year in the Sudan, and certainly it left nothing to be desired so far as appearance and flavour go.

Mosquitoes. Only a few more or less general papers will be here considered, as these insects, so far as the Sudan is concerned, form the subject of a special paper by Mr. Theobald. (*See* Third Report)

The longevity of mosquitoes has been the subject of enquiry, especially as regards *Stegomyia fasciata*, or rather *Calopus*, as the carrier of yellow fever has been re-named. Finlay [2]* of Cuba, has found that when an infected *Stegomyia* is not allowed to bite and get its due nourishment of warm blood, it is prevented from laying its normal complement of eggs, and may live as long as four or five months. This is important in connection with the outbreak of yellow fever.

A very important and practical report is that by Smith,[3] of New Jersey. A great deal of information is given regarding the habits of mosquitoes, both *imagines* and larvæ. The latter are stated to have apparently little or no influence in purifying the waters they inhabit. Their food consists chiefly of the spores of algæ and other vegetable matter. One interesting point discussed is as to whether blood is a necessary food to enable a female mosquito to mature her eggs. "As to this," says Smith, "there is still considerable doubt," and continues :—

> It is certainly proved within my own experience that *Culex pipiens* may oviposit without food other than that which could be found under the net covering a common wooden pail in which the parent developed. It is certain, too, that there are long stretches of salt marsh breeding areas on the New Jersey Coast, where mosquitoes occur by the million, where the foot of man does not touch once a year, where no warm-blooded things save a few birds abide, and where blood is absolutely unattainable. Of course, a large percentage of these salt marsh breeders migrates inland and feeds bountifully; but none of these migrants seem to be fertile and the blood food produces no developing ovaries. On the other hand, the vast majority of specimens in which ovaries are found to be well developed showed traces of blood food in the stomach. This statement should be qualified, however, so as to apply to *C. sollicitans* only; in *C. cantator* there is usually no trace of food observable to the naked eye when ovaries are fully developed.
>
> Very few direct experiments were made on this point with other than the species above mentioned; but published records indicate that in captivity some species will not develop eggs or lay them until after a meal of blood. Whether that would hold equally true of the same species, under entirely natural conditions, may be considered questionable. Incidentally it may be said that not only are all warm-blooded animals and all birds attacked by mosquitoes, but the reptiles also, where they afford an opening. On the whole, the balance of evidence is perhaps against the idea that blood is a necessity for egg development. This is further indicated by the fact that *Anopheles* goes into hibernation without having fed, and that there are few records of biting early in the season, before these hibernating forms lay their eggs to produce the first brood of larvæ.
>
> "That mosquitoes feed upon vegetable juices as well as blood is certain. As to the males, it must be so, if they feed at all; for their mouth structures are not adapted to puncture the skin or to suck blood. Females have been observed along with nectar-sucking males, but seemingly these abandoned the vegetable food readily when the animal odour advised them of something more to their taste."

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[1] *Journal of Tropical Medicine and Hygiene*, 2nd March, 1908, Vol. XI.

[2] Finlay, C. Quoted in *British Medical Journal*, September 14th, 1907.

[3] Smith, J. B. (1904), "Report of the New Jersey State Agricultural Exp. Station on Mosquitoes."

* Article not consulted in the original.

Mosquitoes *—continued*

The questions of hibernation and migration are discussed, but the former at least is not of interest to us in the Sudan, where there is no need for such a stage in the life-history of the insect.

The chapter on natural enemies is full of interest. As regards the adults, one notes that they are taken by spiders and by numerous predatory insects, by frogs, toads, lizards, bats and birds. The myth of the dragon-fly as a great mosquito destroyer is, however, exploded. Birds, especially night birds, are effective. It is said that the tiny red parasitic mites, which infest mosquitoes, and which have been found in the Sudan and Uganda, serve to weaken the insect and possibly to shorten its life; but it is admitted that little is known with certainty regarding the life-history of these minute parasites.

The round worm *Agamomermis culicis*, is stated to be a much more effective enemy, but, so far as is known, it only affects one species (*Culex sollicitans*), being found in the abdominal cavity.

The presence of gregarines in mosquito larvæ in India is mentioned, as is also the infestation of the *imagines* by filamentous phytoparasites, by pathogenic yeasts, by Acarines (external parasites), by Crithidia, sporozoa and even minute trematodes.

The enemies of the larvæ, apart from weather conditions and disease, consist of fish, to which a special chapter is devoted—and this is a line of work which should be taken up with reference to the Nile Fish now that Boulenger's[1] treatise is available, and these can readily be identified.

Birds are mentioned. In the Sudan one may note that various species of water-wagtail are very effective. Then follows a long list—the *Dytiscids* or diving beetles, the whirligig beetles or *Gyrinidæ*, the water-boatman and the water-strider, the water-scorpion and others. Many of these are figured. The larvæ of dragon-flies, it is noted, are bottom feeders and are of little use. I have experimented with them and the larvæ of *C. fatigans*, and can confirm this statement, though occasionally they did devour larvæ.

Finally, the cannibalistic habits of the larvæ receive attention, together with the influence of plant enemies, such as duckweed (*Lemna*) and *Spirogyra*.

There is, indeed, no book with which one is acquainted that contains such a mass of interesting details as this valuable and practical report.

In discussing larvicides, it recommends Phinotas oil, a preparation made in New York, as being most effective, but it is so deadly that it cannot be used where fish exist. Common kerosene, crude petroleum and chloronaphtholeum are mentioned, and the places suitable for treatment by them are indicated. Chloride of lime is said to be more active than lime itself. Even so small a quantity as fourteen grains in one quart of water will kill all stages except pupæ ready to transform in a few hours. It makes excellent material for treating gutters and drainage ditches. For this purpose it should be finely divided, and should be spread or dusted freely over the surface. I may say I have tried this in river pools in Khartoum. It is true that it kills any larvæ present, but it soon sinks to the bottom, and the pools tend to become speedily re-infected. As Smith says:—

> There are really two different types of materials that are used as larvicides; those that make a film on the surface through which the larvæ and pupæ cannot safely penetrate to breathe, and those which mix with the water itself and either poison it or destroy the food of the larva. Each type has its advantages and its limitations. The advantage of the oils that form surface films and do not mix with the water is that it is easy to determine the amount needed, and that for a given area it is always the same whether the pool be deep or shallow. Another point in their favour is that the action is as positive against pupæ as against larvæ. The disadvantage is the ease with which a film is destroyed and the short time that is needed to form a good breeding place after the application is once made.
>
> The advantage of those materials that actually poison the water is that the latter is rendered unfit for larvæ so long as the poison remains or is not materially diluted. The disadvantage is that, as a rule, they do not act promptly or at all on the pupæ.
>
> *Phinotas oil* and the *soluble crude oil* belong to both types and should be, theoretically, the best of all. But both are too destructive to aquatic life generally, where such exists, and are not as active disinfectants as some of the Cresol preparations for gutters, etc., besides being dirty.
>
> Conditions vary, and no two can be dealt with in just the same manner. There are plenty of tools to work with, and that should be selected which fits best.

Of repellents, oil of citronello *(sic)* is said to be quite effective, so that, in this particular, American mosquitoes appear to differ from their African relatives.

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[1] Boulenger, G. B. (London, 1907), "The Fishes of the Nile."

squitoes -continued

Felt,[1] in a somewhat similar report on the mosquitoes of New York State, describes the fungus diseases attacking mosquitoes. These are *Entomophthora spærosperma* (Fersn.), which attacks other insects as well, *Empusa culicis* (Braun), like the fungus of house-flies, and, possibly, *Empusa papilata*. A new species of *Entomophthora* has also been described. Attempts made to spread the disease caused by it failed.

McWeeney[2] deals with Schaudinn's work. The latter investigated the œsophageal diverticula of *Culex pipiens*.

> These become distended with gas and may be termed "gas-bags." The gas is carbon dioxide which is evolved by a sort of yeast-like fungus always present in the insect's stomach from traces of glucose present in blood, or—much more abundantly—from glucose present in the plant juices which the insect occasionally sucks. At the commencement of the act of suction, when the insect has its proboscis buried in the skin of its victim, its body undergoes one or more violent contractions which eject the contents of its foregut and "gas bags" into the skin. These contents comprise gas, saliva and whatever particulate matter is present—viz., yeast cells and sporozöites. Schaudinn looks upon this contraction as a sort of dyspnœal effort due to the entrance into its trachæ of the CO_2-laden air which bathes the skin. He succeeded in producing the contraction artificially by placing an infected culex on a cupped slide, with its proboscis in a drop of glycerin under a cover-glass, and its body projecting over the hollow. In this he evolved some CO_2 from a fragment of chalk and a droplet of acid, and he observed that the gnat's body underwent a violent contraction, which had the effect of expelling into the glycerin its contained gas, yeast cells and sporozöites. Viewed teleologically, the effect of the injected CO_2 would be to paralyse the thrombocytes and in other ways to delay the coagulation of the blood. The hyperæmia and pain caused by the bite he considers to be due to the enzyme of the yeast cells. He dissected out the "gas-bags" and pushed them into a fine puncture in his own skin, with the result that the typical swelling, redness, and itching came on at once. The salivary glands he found quite inoperative. The effect of the "gas-bags" appears to depend on the quantity of yeast cells contained in them, for it was much more marked when the yeast had been allowed to proliferate actively as the result of feeding the gnat on plant juice. The fungus is not a true yeast, but a yeast-like stage in the life-history of one of the *Entomophthoræ*, fungi with which we are all acquainted in the shape of the well-known *Empusa muscæ*, which kills flies in the fall of the year and causes their bodies to adhere to the window pane surrounded by a white cloud of ejected conidia.

In view of the discovery of a new species of Sudan mosquito, *C. salus*, breeding in sea-water, the observations of Foley and Yvernault[3] are of interest. They found in Algeria that an anopheline, *Pyretophorus chaudoyei*, was able to breed out in very saline waters. The same, they note, as being true of *Anopheles vagus* found in the Dutch East Indies.

Some notes on applications for allaying the pain of mosquito bites may be useful. A mixture one has seen highly recommended consists of a half-pint 1 in 20 carbolic acid and 4 oz. No. 4711 Eau de cologne.

Schill[4]* advises applying a paste or saturated solution of bicarbonate of sodium to the bitten part; while as a repellent, 50 per cent. alcoholic solution of thymol may be tried.

Joly's mixture is as follows:—

Liq. formaldehyd (40 per cent.),	℥ iv.
Xylol,	℥ iss.
Acetoni,	ʒ i.
Balsam-canaden.,	gr. xv.
Ol. citronellæ,	*q.s.*

Before applying, shake the mixture and touch the bitten part with the end of the wetted cork or small piece of cotton wool, and then allow the fluid to dry on the skin.

As regards measures for destroying the *imagines*, one has been accustomed to use the sulphur squibs advocated by Giles,[5] but they merely stupefy the insects which have afterwards to be killed. An Indian remedy consists of a tablespoonful of potassium nitrate 1 part, powdered chrysanthemum 4 parts, and powdered nard root (*Nardus indica*, or Spikenard) 4 parts.

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[1] Felt, E. P., "Mosquitoes or Culividæ of New York State." Albany, 1904.

[2] McWeeney, E. J. (March 25th, 1905), "On the Relation of the Parasitic Protozoa to Each Other and to Human Disease." *Lancet*, Vol. I.

[3] Foley, F. H., and Yvernault, A. (March 11th, 1908), "Anophelines dans d'eau Salée." *Bull. Soc. Path. Exot.*, Vol. I.

[4] Schill, "Mosquito Bites." Quoted in *Journal of Tropical Medicine*, November 1st, 1906, Vol. IX.

[5] Giles, G. C., "Gnats or Mosquitoes." London, 1902,

* Article not consulted in the original.

Mosquitoes —*continued*

Kendall[1]* tabulates some useful facts regarding fumigation :—

1. Preparation of the house: disturb the apartments as little as possible; stop up all openings; have the door guarded by a canvas curtain.

2. To fumigate: for each 1000 cubic feet of air space, 2 lb. of sulphur, or 2 lb. to 4 lb. pyrethrum, are placed in a pot and set alight.

3. After a few hours the house is opened up, the mosquitoes swept up with a damp broom, the paper and paste used in stopping up cracks removed. Of the several fumigants, sulphur is the most convenient for use. Pyrethrum, also known under the names of "Bubach," Persian insect powder and Dalmatian powder, is used, but the powder should be that obtained from unexpanded flowers, and not the adulterated varieties frequently offered for sale.

Campho-phenique, called also Mimm's mixture, consists of a mixture of equal weights of camphor and (95 per cent.) carbolic acid, and has proved a fairly reliable culicide. Four ounces of the mixture is placed on shallow pans for each 1000 cubic feet of air space, and subjected to the heat of an alcohol lamp.

Of the three fumigants mentioned, campho-phenique has the advantage of being cheap, efficient and non-objectionable. Sulphur is efficient, but proves injurious to fittings and fabrics. Pyrethrum is unreliable and causes darkening of light-coloured paint and similar substances.

Several other fumigants have been experimented with. Concerning these it is stated: Hydrocyanic acid is dangerous to human beings, owing to its poisonous fumes; chlorine gas has the disadvantage of bleaching fabrics; carbon bisulphide is dangerous owing to its inflammability; Jimson weed or stramonium is unreliable; formaldehyde is an unsatisfactory insecticide, although so potent a bactericide.

Other papers which may be noted are those of the brothers Ross,[2] on their automatic oiler adopted for cesspools in Cairo. It is fashioned from an old paraffin tin, and is both cheap and ingenious. It is both described and illustrated, and may be conceivably used elsewhere with advantage.

Another apparently ingenious French contrivance is the mosquito trap invented by Blin,[3] which is really an artificial refuge into which the insects penetrate and from which they cannot escape. These *trous-pièges*, as they have been called, require to have their efficacy tested, but may be of value in certain regions. Considerations of space and time forbid their being here described.

Mycetoma. This disease is by no means rare in the Sudan, and is one which merits careful investigation, for, if its precise etiology could only be determined, preventive measures against it might be put in force. I believe I have seen at least three types of madura foot, but unfortunately there has not been time to study the question fully, and indeed the whole matter is still the subject of controversy. Of recent work none is more important than that of Brumpt,[4] who has established a special classification and distinguishes no less than eight different varieties of the disease. Of these, two are due to a species of *Discomyces* and two to *Aspergillus*. Of the remaining four, less is known. They are probably all due to species of *Aspergillus*, but, in the absence of cultural proof, they have been placed in two groups, named respectively *Indiella* (unpigmented septate species) and *Madurella* (pigmented species).

The fungi concerned, not only present resisting forms like sclerotia and chlamydospores, but also characteristic spore apparatus.

As Manson[5] gives Brumpt's classification in some detail, one need only refer briefly to it here. The species are :—

1. Actinomycotic Mycetoma due to *Discomyces bovis*, the ray fungus.

2. Vincent's White Mycetoma caused by *Discomyces maduræ*.

This runs a slow course, does not destroy bone, and does not directly affect the general health. The grains vary in size from that of a pin's head to that of a pea. They are yellowish-white, have a mulberry-like surface, and are soft. They grow by throwing out radiating fungus threads, and present a typical appearance. Spread takes place

[1] Kendall, A. I. (December 1st, 1906), "Experiments on Practical Culicidæ Fumigation." *Journal of Tropical Medicine*, Vol. IX.

[2] Ross, E. H., and Ross, H. C. (June 15th, 1907), "An Automatic Oiler for the Destruction and Prevention of Mosquito Larvæ in Cesspools and other Collections of Water." *Annals of Tropical Medicine and Parasitology*, Series T. M., Vol. I., No. 2.

[3] Blin, G. (February 12th, 1908), "Destruction des Moustiques par le procédé des trous-pièges." *Bull. Soc. Path. Exot.*, Vol. 1.

[4] Brumpt, E. (November 25th, 1906), "Les Mycétomes." *Arch. de Parasit.*, t. X., No. 4.

[5] Manson, Sir P., "Tropical Diseases." 4th Edition. London, 1907.

* Article not consulted in the original.

Mycetoma *—continued*

by the detachment of small shoots which grow on their own account. The grains are found in cavities surrounded by inflammatory tissue infiltrated with leucocytes and sometimes giant cells. This is one of the forms which, I believe, exists in the Sudan.

3. Nicolle's White Mycetoma caused by *Aspergillus nidulans*. The grains are more or less spherical, and present a smooth surface. This fungus destroys bone, and only one case is yet on record. It occurred in Tunis, and showed spore formation.

4. Bouffard's Black Mycetoma caused by *Aspergillus bouffardi*. The grains are black, and vary in size from that of a pin's head to that of No. 1 shot. They have a mulberry surface, which is smooth and glossy. They are elastic, but break when pressed. The grain consists of a coiled-up mass, which unfolds when macerated in water, displaying a densely felted mycelium. The grains occur in the cellular tissue always singly and within small cavities. Large giant cells form a feature of the growth. Curettage may cure this form, which may, however, extend by way of the lymphatics.

5. Classic Black Mycetoma caused by *Madurella mycetomi*. The grains are dark brown or black. Each measures 1 to 2 mm. in diameter, and is hard and brittle. The surface is irregular and more or less "spiked." The grain is composed of white threads and cement substance, and, after a period of active growth characterised by the formation of chlamydospores, passes into a resting stage and becomes a sclerotium, in which form it is eliminated. This type is very destructive, forming large and sometimes fungating tumours, and is by far the commonest form in the Sudan. I have seen it affecting the exterior of the knee-joint and the hand, and noted that it may spread by way of the lymphatics.

6. Brumpt's White Mycetoma caused by *Indiella mansoni*, an Indian form. The grains are hard, white, small and resistant.

7. Reynier's White Mycetoma caused by *Indiella reynieri*, and described from a case of madura foot in a man who had never been out of France. The grains, which are soft and white, were found rolled up like the excrement of earth-worms.

8. Bouffard's White Mycetoma, caused by *Indiella somaliensis*. Single grains are small and smooth, and consist of a mycelium which at its earliest stage is always found in a giant cell. The grains vary in colour from white to reddish-yellow, and are found clustered together like fish-roe in the sinuses. This fungus is most destructive, attacking bone and producing sclerosis. I am inclined to think that I have seen one case of this form (pink mycetoma), a specimen of which is in the laboratories' museum.

Brumpt's paper is very well illustrated, and amongst his conclusions we note that—

(1) it would seem that for the production of mycetoma there must be, both on the part of the host and parasite, conditions not easily obtainable in Nature, otherwise the number of cases would be enormous, owing to the apparent facilities for spread amongst the bare-footed natives of those countries chiefly affected; (2) on account of the feeble resistance of the conidial spores in the tissues and the slight success which has attended the experimental subcutaneous injection of spores, it would seem that the fungus must be inoculated into the tissues in a form better adapted to resist destruction than filaments given off by the conidia; (3) the giant and epithelioid cells appear to play a part in the nourishment of the young parasite, and, what is remarkable, the macrophages, which should defend the threatened organism, actually act as disseminators of the fungus and so aid its spread; (4) the diagnosis is to be made from the grain which is characteristic. This is not the case as regards the tumour masses, the appearance of which varies according to the age of the lesion; (5) no doubt other forms will be found to exist.

Musgrave and Clegg,[1] in a paper on the etiology of mycetoma, whilst acknowledging that Brumpt's work is very exhaustive, conclude that he has made his classification on insufficient data. This may be so, but it is likely to be helpful, and marks a step in advance. Musgrave and Clegg describe the disease in the Philippines and claim to have isolated a new parasite, *Streptothrix freeri*, from a single case. They are of opinion that all types of mycetoma are due to Streptothrix infections, albeit it is not yet settled whether more than one species plays a part in the disease. Their paper is chiefly of value because of the review of the literature which it contains and the extensive bibliography appended to it.

Vincent[2]* applies the name *Madura* to the variety with white grains due to *Discomyces maduræ*, and the name *Mycetoma* to the variety with black grains caused by a fungus with a septate mycelium *(Madurella mycetomi)*. Resistance to staining is regarded as an indication of degeneration, or loss of vitality in the fungus, due to long duration of a case.

[1] Musgrave, W. E., and Clegg, M. T. (December, 1907), "The Etiology of Mycetoma." *Philippine Journal of Science*, Vol. II., B. Med. Sciences.

[2] Vincent (July 28th, 1906). *C. R. Soc. Biol.*, t. LXI.

* Article not consulted in the original.

Pinoy[1]* has succeeded in infecting the foot of a pigeon with a white mycetomatous growth of human origin. Strange to say, the grains in the pigeon were black. The fungus was first cultivated in sweetened bouillon under anærobic conditions. **Mycetoma** —*continued*

Myiasis or, more correctly, *Myiosis* (Gould). This subject, as far as the human being is concerned, is of special interest to medical officers in the Southern Sudan, and especially in the Bahr-El-Ghazal Province. Some facts have already been collected regarding myiasis in the Sudan (*vide* Second Report), but there is no doubt that a great deal remains to be discovered, and Mr. King is paying special attention to this subject.

Wellman[2] gives the following list of diptera known to cause human myiasis :—

Œstridæ

Gastrophilus. Horses and man.

Hypoderma. H. bovis, man. *H. diana*, deer and man.

Dermatobia. The larvæ of *D. cyaniventris* is the "Ver Macaque" of tropical America, and in man causes painful boils, occasionally attacking the eyes; also *Hypoderma bovis*, reported by Scheube.

Sarcophagidæ

Sarcophaga. S. carnaria, S. magnifica and *S. ruficornis* occasionally deposit their larvæ in wounds of man (India). A species of this genus (*S. sp.* near *regularis*) is the fly used in the experiment detailed in this paper.

Sarcophila. Man and animals.

Auchmeromyia. A. luteola, in Angola, and another species (*A. depressa*), cause cutaneous myiasis in Natal (but *vide infra*).

Ochromyia. The larvæ of *O. anthropophaga* is the "Ver du Cayor," which in Senegal produces cutaneous inflammation and swellings.

Muscidæ

Musca. Larvæ of *Musca sps.* occasionally are passed in fæces or found in wounds.

Calliphora. In intestines of man and animals.

Compsomyia. The larvæ of *C. macellaria* is the "Screw-worm" of tropical America.

Lucilia. L. sericata is the cause of "maggot" in sheep. The larvæ of several species of *Lucilia* have been detected in wounds and ulcers in man and animals.

Anthomyidæ

Anthomyia. The larvæ of *A. canicularis* not seldom get into the stomach and intestines of man, through eating raw vegetables.

Hydrotæa. In the fæces of human beings.

Homalomyia. In the intestines of man, being passed alive in the fæces. Osler gives a case of infection by *H. scalaris* in Louisiana.

Hylemyia. In human excreta.

In a later paper[3] he supplements this, describing a case of intestinal infection by the larvæ of *Anthomyia desjardensii*, the symptoms being abdominal tenderness, foul breath and nervous distress. In Angola he has noticed myiasis produced by *Sarcophaga africa, S. albofasciata, S. sp. incert, Auchmeromyia luteola* and an unknown Muscid larva.

Gedoelst[4] has a contribution to our knowledge of the larvæ of flies causing myiasis in Africa. The principal, he says, are :—

1. The Ver du Cayor, the larva of *Ochromyia anthropophaga.*
2. The larva of Natal, which he says is probably that of *Bengalia depressa* (but *vide infra*).
3. The larva of *Cordylobia anthropophaga*, Grünberg.
4. The larva of Lund found in the Congo Free State. The last is possibly the now well-known Congo Floor maggot, the larva of *Auchmeromyia luteola.*

Recent investigation goes to show that there is no definite evidence incriminating *Bengalia depressa.* Its larvæhave been co nfounded with those of *Cordylobia anthropophaga*, apparently a much more important fly. This is noted by Austen[5] in a paper on this fly, which on the West Coast of Africa is called the Tumbu fly. It occurs in the Southern

[1] Pinoy, E. (1907). *C. R. Acad. Sciences*, t. CXLIII.

[2] Wellman, F. C. (June 15th, 1906), "Experimental Myiasis in Goats, with a Study of the Life Cycle of the Fly used in the Experiments, and a List of some similar noxious Diptera." *Journal of Tropical Medicine*, Vol. IX.

[3] Wellman, F. C. (June 1st, 1907), "Intestinal Myiasis in Angola." *Journal of Tropical Medicine and Hygiene*, Vol. X.

[4] Gedoelst, L. (July 1st, 1905), "Contribution à l'étude des larves cuticoles de Muscides Africaines." *Arch. de Parasit.*, t. IX., No. 4.

[5] Austen, E. E. (January, 1908), "The Tumbu Fly (*Cordylobia anthropophaga*, Grünberg)." *Journal of the Royal Army Medical Corps.*

* Article not consulted in the original.

Myiasis— *continued*

Sudan, and Mr. King deals with it in his section of the Third Report. In the same number of the journal there is an account of the fly from a medical standpoint, written by Major Smith. He says :—

> The larva of the Tumbu fly burrows beneath the skin of human beings and other animals, and becomes stationary. The cavity in which it lives is not cut off from the external air; an opening is always left, and in or near this the posterior end of the maggot lies. When mature it drops out, burrows into the ground and becomes a pupa.

Experimenting with immature larvæ, Smith found the flies to appear on the sixteenth and seventeenth days.

> In the human being the appearance of the lesion produced by the larva is that of a raised, reddish patch; on a clean washed skin it looks something like an urticarial wheal. At some part of this swelling will be seen a tiny opening, or a moist spot, perhaps a blackish mark, according to how much, if any, of the larva is presenting at the opening, and to the stage of growth. In some cases, where the skin has not been washed, pus may have exuded and scabbed around the orifice, so that the appearance is that of a broken boil. There is intense itching in and around the spot. Strong pressure towards the opening forces the larva out easily enough, so that in adults familiar with the fly the larva does not get a chance to grow very big, unless it happens to be in a part where the sufferer cannot see what is wrong. In neglected children and helpless people the larva is able to grow to its full size. In such cases there is usually suppuration in the cavity, and it is common, on ejecting the intruder, to see a bleb of pus follow it out. No serious results are known to ensue, but an avenue is provided for the entry of germs.

"Tumbu" have been found in men, dogs, monkeys, rats and imported guinea pigs.

In Europeans, who are not commonly attacked, the scrotum is a usual site, in negro natives the head, but no part of the body is exempt. Babies are often affected. The ordinary and commonest mode of infection is undoubtedly from the ground.

Blenkinsop[1] also has some notes on this disease as it occurs in Sierra Leone. He mentions that multiple infection is common, and that in Europeans the upper parts of the thigh and the buttock are favourite sites for the larvæ. This favours the view that they are often acquired at the latrine. He describes the lesions as resembling furunculi and being at times extremely painful. Examination of the central area of the inflammatory zone reveals the presence of some black matter, the excrement of the larva immediately beneath the skin surrounding the minute breathing aperture. On pressure over this spot intense pain results. In ordinary cases the pain is paroxysmal. Irritation may lead to abscess formation. Blenkinsop gives the following as serving to differentiate the lesions from boils :—

> (1) The presence of the black excrement; (2) the pain caused by gentle and continuous pressure on the breathing aperture; (3) the paroxysmal character of the pain, which is generally unaccompanied by throbbing. As regards treatment, the larva can usually be easily removed entire with the point of a surgical needle, and the small wound should then be washed out and dressed antiseptically. If suppuration has occurred, free incision is necessary.

Blenkinsop notes that a plaster of soap and sugar caused the larvæ to appear at the surface in less than twelve hours, and considers that this result was probably due to blocking of the breathing aperture with the plaster.

Lelean[2] has an interesting article on Myiasis, and we quote here his notes on treatment :—

> In a *gusano* worm the larva of a "mosquito-like" fly found in Guatemala, the natives (1) occlude the orifice of the cavity in which the larva is contained by a piece of stamp paper; the air-breathing parasite, being thus asphyxiated, can be expressed; (2) cover the aperture with a tobacco-leaf, the nicotine poisoning the grub.
>
> Dr. Folker uses a hypodermic of chloroform which so paralyses the larvæ that he has by this means expressed as many as fourteen in less than two minutes, a velocity which commands respect. The *Dermatobia noxialis* (screw-worm) is killed in the frontal sinus by carbolic injections of 2 per cent. solution.
>
> In the auditory meatus the larvæ often cause so much tenderness as to make mechanical extraction impossible. A little calomel blown into the meatus is said to cause their death and spontaneous extrusion.
>
> Lieutenant-Colonel J. Smith, writing of Indian experience, found maggots ingested in mangoes most difficult to dislodge, a fact not to be wondered at when they survived five minutes' immersion in pure carbolic acid. One patient passed from fifty to a hundred larvæ daily for twelve months, and intensely feared their eating through the intestine. One case was cured by enemata terebinthinæ; another was on butea frondosa, and in a third—parasiticides having no effect—scybala were produced by opium, and the embedded maggots came away by subsequent use of purgatives and enemata.
>
> Finally, in huts infested by diptera, if cones of dried pyrethrum powder be burnt, the flies fall stupefied to the floor, whence they can be collected and burnt.

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[1] Austen, E. E. (January, 1908), "The Tumbu Fly (*Cordylobia anthropophaga*, Grünberg)." *Journal of the Royal Army Medical Corps.*

[2] Lelean, P. S. (January 30th, 1904), "Notes on Myiasis." *British Medical Journal*, Vol. I.

Commenting on these notes, Younge[1] recommends the following method for getting rid of subcutaneous larvæ :— **Myiasis—** *continu*

The tip of an ordinary probe is lightly smeared with vaseline and pressed on to a little calomel so as to take up about 1 grain of the drug. It is then passed into the cavity containing the larvæ and gently moved round it. The calomel kills the larvæ in a few minutes. They can then be removed by gentle pressure, or, better still, by syringing the cavity which contains them with a little warm boracic lotion.

The other drugs, such as turpentine, which have been recommended for destroying maggots, are uncertain, irritating and very painful. On the other hand, calomel acts rapidly, with certainty, and without causing the slightest pain or discomfort. It also seems to destroy or neutralise the excretory products of the larvæ, which are often sufficiently irritating to excite considerable local inflammation and high fever.

Calomel is also fatal to most of the lower forms of life, and I have used it successfully to get rid of a leech which had accidently entered the nasal fossa.

Blankmeyer[2*] has described a case of infection with the larvæ of *Anthomyia canalicularis*. The symptoms consisted of abdominal pain and distension, with bloody diarrhœa, followed by constipation. Treatment of many kinds was tried, but after the patient had eaten raw pumpkin seeds on an empty stomach for three days, a saline purge was given, when from 1000 to 1500 larvæ were expelled in a bulky stool. A few continued to come away for several weeks.

It used to be believed that *Hypoderma bovis* was able to pierce the skin with its ovipositor and deposit its eggs directly in the subcutaneous tissue where they underwent development. Jost,[3*] however, has recently proved that—

The egg is fixed by the fly on the hair of the host, and is introduced into the mouth by licking, probably still unhatched. The young larvæ are only to be found in the lower part of the œsophagus and commencement of the stomach. Thence they make their way into the submucosa of the œsophagus and travel under the pleura or peritoneum to the sides of the vertebral column, generally by way of the mediastinum and pillars of the diaphragm, and the capsule of the kidneys, and follow the vessels and nerves through the inter-vertebral notches, where they may be found from December to March. From thence they pass out into the inter-muscular planes of the dorsal region, and so reach the skin, from which situation they escape after about a month, being in the meantime enclosed in a capsule formed by the host. The larva undergoes two ecdyses, becomes a pupa, and this finally gives exit to the imago.

The Sergents[4] have brought to light a form of human myiasis occurring in Algeria amongst Kabyl shepherds. It is due to *Œstrus ovis*, a sheep parasite, called locally Thim'ni, as is also the disease it produces. It deposits its ova while in flight without settling, upon the eyes, the nostrils or the lips of shepherds, especially those who have eaten of the fresh sheep or goats' cheese. The condition is also found in dogs fed on cheese.

Irritation and inflammation is produced at the site of deposition, great pain if the nose be attacked, together with frontal headache and nasal discharge. In the case of the lips, the inflammation may spread to the throat. Tobacco is the best cure, used as snuff or smoke for the nasal condition, or as an infusion in the form of a gargle for the throat.

Shattock[5] records a case of intestinal myiasis due to the larvæ of *Eristalis tenax*. This is a dipterous fly which produces larvæ as large as tadpoles and furnished with a respiratory proboscis, hence the name "rat-tailed larvæ." A similar kind of fly, *Helophilus trivittatus*, is described in our Second Report, but there is no record of its producing myiasis.

Onyalai. This is a disease of Portuguese West Africa, which has been described in two papers by Massey[6 7] and one by Wellman.[8] As it is quite possible that it exists in the

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[1] Younge, G. H. (February 13th, 1904), "The Treatment of Myiasis." *British Medical Journal*, Vol. I.

[2] Blankmeyer, H. C. (May 4th, 1907). *Journal American Medical Association.*

[3] Jost, H. (1907), "The Development of the larvæ of Hypoderma bovis," de *Geer. Zeit. für Wiss. Zool.* Bd. LXXXVI. Quoted in *Journal of Tropical Medicine and Hygiene*, November 15th, 1907, Vol. X.

[4] Sergent, E., and Sergent, E. (May 25th, 1907), "La Thim' ni, myiase humaine d'Algérie, causée par *Œstrus ovis*," L. *Ann. de l'Instut. Pasteur*, Vol. XXI., No. 5.

[5] Shattock, G. S. (March 28th, 1908), Larvæ of *Eristalis Tenax* passed by the Bowel. Report of Meeting of Roy. Soc. of Med. Path. Sec. *Lancet*, Vol. I.

[6] Massey, A. Y. (September 1st, 1904), "Onyalai, a Disease of Central Africa." *Journal of Tropical Medicine and Hygiene*, Vol. VII.

[7] Massey, A. Y. (April 1st, 1907), "Onyalai, a Disease of Central Africa." *Journal of Tropical Medicine and Hygiene*, Vol. X.

[8] Wellman, F. C. (April 15th, 1908), "A Fatal Case of Onyalai, with Remarks on the History, etc." *Journal of Tropical Medicine and Hygiene*, Vol. XI.

* Article not consulted in the original.

lai—
ntinued

more tropical and humid regions of the Sudan, such as the Bahr-El-Ghazal Province, it is perhaps well to direct attention to it, the more so that Feldman has seen a somewhat similar disease in East Africa called "Edjuo," and Mense, on the Congo, met with a condition exhibiting some of the symptoms of "Onyalai" and termed "Kafindo" by the Unyamwezi people.

The etiology is quite obscure. Wellman seems to have proved that it has nothing to do with malaria, trypanosomiasis, Schönlein's disease, Henoch's purpura, Purpura hæmorrhagica, accidental or intentional poisoning or snake bite, though it may resemble the effects produced by the bite of the puff-adder (*Clotho arietans*, Gray).

Wellman, indeed, considers the disease to be a specific entity, an acute infectious disease, the cause of which is as yet undetermined.

The account given of the clinical features is here reproduced :—

Most of my cases were attacked suddenly. Lassitude and a sort of dazed appearance was generally marked. In some cases the parotid glands were tender to the touch. The eyes appeared heavy and sometimes reddened. In most instances the tongue was swollen and painful. A slight temperature was noted in about a third of the cases. I have had patients complain of numbness and of pain in various parts of the body. One man who had bloody diarrhœa suffered much from colicky pains. The appetite is usually poor. The bullæ may appear in the mouth, pharynx, œsophagus, stomach and bowels. Vomiting of blood is not rare. The skin also usually shows lesions. The genito-urinary system was affected in three of my cases. Hematuria was a prominent symptom in all of these. When the process occurs in the cranial cavity various symptoms of profound central disturbance are set up. Three cases of this kind have come to my attention. One of these was a young woman in rude health. She was laughing and playing on the evening of her attack. Suddenly she complained of being tired, and in an hour or so bullæ appeared in her mouth. She steadily grew more depressed, and died about eight o'clock the next morning, with all the symptoms of cerebral hæmorrhage, *i.e.* loss of consciousness, inactive, dilated pupils, slow, noisy, stertorous breathing, etc. The pulse was very slow and increased in tension. Another fatal case in the same house, a strong young man being the victim, presented almost exactly the same symptoms. A third fatal case showed no vesicles, either in the mouth or in the skin. The fæces and urine likewise contained no blood. Another case which I have also referred to this disease likewise showed no vesicles, but died from the effect of what I believe to be the same process in the liver, spleen and pancreas. (These last two cases were shown microscopically not to be pernicious malaria.) The superficial bullæ, when present, are characteristic. They range from the size of a split pea to several inches in diameter. The larger ones are irregular in outline, and are often umbilicated. They are deep, and involve the corium or submucous structure. There seems to take place an extensive histolysis, only the fibrous elements persisting. These appear as trabeculæ, the interstices of which are filled with partially coagulated and otherwise altered blood which shows dark under the skin or mucous membrane. The red corpuscles, however, are not all disintegrated, and may be seen under the microscope in the oozing from the blebs, and also in the fæces, urine, saliva, etc., according to the situation of the lesions. In one or two cases the bullæ were very small, and not numerous, and had the patients not directed attention to them would probably have been overlooked.

The Congo disease described by Mense[1] is characterised by depression, malaise, headache, reddened conjunctivæ, numbness, swelling of the tongue, loss of appetite and occasional bloody diarrhœa, dyspnœa, cardiac disturbances, etc., all of which have been noted in "Onyalai." Bullæ, however, were not observed.

The prognosis of "Onyalai" is still an unknown quantity. The malignancy of the disease appears to vary greatly. Natives often regard it as very fatal. No treatment seems of much use, except possibly arsenic in large doses.

Information is desired regarding the possible occurrence of this interesting condition within the confines of the Sudan.

Oriental Sore. So far as one can find out, this condition does not exist in the Sudan. Colonel Hunter, P.M.O., tells me he has never seen it. At the same time, it is one of much interest, and as it is associated with the presence of parasites identical with the Leishman-Donovan body, and as kala-azar occurs in this country, it is necessary to consider recent work upon it.

In a recent important discussion on the subject, Manson[2] points out that the disease has the peculiarity of being protective against itself. He also alludes to the difficulty in stating the duration of the incubation period, which is sometimes short, but frequently runs into months. Nearly always an exposed part of the body is affected, and Manson suggests that the disease is inserted by some animal which attacks these parts. The bug, flea and similar insects are therefore excluded. The mosquito and various flies would, however, probably be effective carriers. The disease is inoculable, but inoculation experiments may fail possibly because the parasites have disappeared from the sore or are in an unhealthy condition or have undergone involution.

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[1] Mense, C. (1906), Handbuch der Tropenkrankheiten, p. 789, Vol. III.

[2] Manson, Sir P. (December 2nd, 1907), "Demonstration of Oriental Sore and its Parasite." *Journal of Tropical Medicine and Hygiene*, Vol. X.

He speaks to the morphological identity of the parasite of Oriental sore and that of kala-azar, and, in order to see if they were specifically identical, he attempted to inoculate a kala-azar patient from a case of Oriental sore. Unfortunately both the test inoculation and the controls failed. Manson believes Oriental sore to be a blood disease, and that if it be cured in one place it will break out in another. If, however, the disease is obtained in the involution stage, pressure and local application may hasten the cure. Low thought the disease might be due to a spirochæta, while Duncan mentioned a case successfully treated by the application of a disc of lead the same size as the sore. Sambon mentioned the liability to recurrence and the outbreak of successive crops of a peculiar eruption after the appearance of the first sore. He also alluded to the fact that the disease, which was one of towns, occurred in dogs, and that in their sores the characteristic parasite existed. Hartigan mentioned that many of the Jews in Hong Kong suffered from the disease in unexposed parts, a fact which Manson explained, as far as the Jews in Baghdad went, by infection during childhood which was the time of life when the body was not generally covered in hot climates. **Oriental Sore—** *continued*

Fremantle regarded the condition as a local infection, not as a general disease. Manson, in reply, stated that as Duncan had applied the lead compress after five months' ineffective treatment a cure might be expected as the disease was exhausted and inclined to heal spontaneously.

Cox[1] has a good paper on the Baghdad boil which is a disease of cities or rather of streets which are not properly laid and scavenged. The only method of prevention is to disinfect or cauterise thoroughly any cut, wound, abrasion or mosquito bite immediately on its occurrence. He describes the minute papule increasing in size and finally becoming the ulcer of which there are two types, the male and female, so-called :—

(1) The male ulcer is oblong in shape, like a date seed, hence the name of "date-mark," with an irregular, undermined edge and indurated margin: it is tender on pressure, with a dry uneven surface, and it is extremely indolent in character. The ulcer either remains stationary in size or it gradually enlarges, sometimes attaining a diameter of two inches: as a rule, the size varies from that of a hazel-nut to an inch in the wider diameter. On reaching its permanent size, the sore retains its characteristic appearance and soon forms a dry pustular scab, which increases in size in successive layers, until it becomes a nodular crust, when it drops off, leaving the raw surface of the ulcer bare, and then the scabbing starts afresh.

(2) The characteristics of the male ulcer apply also to the female ulcer, and the only difference is that the latter, instead of forming a dry scab, is forever discharging a pale yellow, watery pus, which adds to the distress of the patient. Both kinds leave a permanent scar.

For treatment in the late stages he recommends strong sulphur ointment (20 per cent.) applied on resin plaster with a layer of wool on the top to graduate the pressure of the bandage : this is applied daily for four or five days until the surface of the ulcer looks clean. Then Unguentum Picis is applied until granulations appear. These are touched with blue stone and an ordinary dressing of boric ointment is applied. Healing occurs in from one to six weeks. Arsenic in medium doses helps the cure.

Donovan[2] suggests that the itch insect, *Sarcoptes scabiei*, may be a vector, as he noticed several sufferers from Oriental sore covered with itch.

The parasite was first seen by Cunningham, but was rediscovered and described by Wright, who named it *Helcosoma tropicum*. His description, method of staining and account of its histology will be found in quotations given in the *Indian Medical Gazette* of August, 1904, and the *Journal of Tropical Medicine*, May 16th, 1904.

Billet[3*] found a case originating at Ismailia and presenting Wright's parasite. He suggests that *Anopheles chaudoyei* may be the carrier, owing to its distribution, especially in Algeria where "Biskra boil" occurs.

A recent paper by Marzinowsky[4] gives a very full bibliography and enters more minutely into the question of treatment than is usually the case. He mentions various caustics and astringents which can be employed followed by dusting powders and finally by sublimate wash, but, considering that the healed sore leaves a permanent scar, he is all in favour of operation under cocaine anæsthesia. In those cases where this is not feasible he thoroughly

[1] Cox, W. H. (February, 1904), "The Baghdad Boil." *Indian Medical Gazette*, Vol. XXXIX.

[2] Donovan, C. (March, 1904), "Delhi Boil." *Indian Medical Gazette*, Vol. XXXIX.

[3] Billet, A. *C. R. Soc. Biol.*, t. LX., p. 1149.

[4] Marzinowsky, E. J. (December 24th, 1907), "Die Orient-beulen und ihre Aetiologie." *Zeit. für Hyg. und Infekt.*, Bd. LVIII., No. 2.

* Article not consulted in the original.

riental ore— *continued*

cleans the sore and uses 10 per cent. ferropyrin solution to stop the bleeding. Thereafter the sore is well moistened with 50 per cent. solution of bimuriate of quinine and collodion dressing applied. The same procedure is repeated daily until complete cure results; at the same time, it is sometimes advisable to inject quinine into the periphery of the sore. By this treatment a complete cure usually results in from 7 to 11 days.

He also cites the recent work of Schulgin, who advocates freezing of the sore with ether. This procedure, tried in 300 cases, yielded very good results.

Two French papers may be cited, though they are mainly of a confirmatory nature, as regards the discovery of Wright's parasite.

Mesnil, Nicolle and Remlinger[1] describe it as discovered in Aleppo button, and Nattan, Larrier and Bussière[2] record its discovery in ten cases of Oriental sore at a place on the Persian Gulf. These last two observers examined the blood of their cases, but failed to find the parasite in the peripheral circulation.

Parasites. Under this comprehensive title one proposes to deal merely with general papers on metazoan parasites and such articles as have for their subject the consideration of new or little-known African forms likely to be encountered in the Sudan. We know very little about the metazoan parasites existing in this country, and yet, being so near Egypt and in such close communication with that "land of worms," as it may almost be called, there must be many species, while in the southern regions rare or unknown forms are certain to exist. It is a branch of study which would well repay attention, but for its proper elucidation a trained helminthologist is a necessity. It may yet be possible to arrange that such an observer should have the use of the Floating Laboratory and devote his time to the human and animal Entozoa of the Sudan. If so, much valuable information may be expected. At the same time Dr. Leiper's paper on the material collected by Dr. Wenyon, adds very considerably to our knowledge.

That the importance of the subject is now fully recognised is shown by a paper of Sambon[3] on the part played by metazoan parasites in tropical pathology.

Dealing first with recently-discovered entozoa, he alludes to *Necator americanus*, and then mentions the new strongyloid of man, *Triodontophorus diminutus*, of American origin, and possibly a cause of tropical anæmia. *Œsophagostomum brumpti* is another Sclerostome, found by Brumpt in the form of six immature females, in cyst-like nodules in the walls of the cæcum and colon of a negro in West Africa.

His remarks as regards the Schistosomidæ have already been partly recorded (*see* "Bilharziosis," *page* 17), but in this paper he mentions the finding by Christophers and Stephens of schistosomum eggs in the urine of a Madras native suffering from hæmaturia. These eggs differ from those of *S. hæmatobium* by their greater length and peculiar spindle-like shape.

Speaking of Looss's work, he mentions the removal of *Opistorchis sinensis*, the Asiatic liver fluke from the genus *Opistorchis* to the new genus *Clonorchis*, and recalls the fact that under the old term two separate species had been confounded. There is, then, a large form *(C. sinensis)* usually present in small numbers and comparatively harmless, and a smaller one *(C. endemicus)* always occurring in large numbers, and distinctly harmful. Both are found in China and Japan, and though their structural differences are very slight, Looss is convinced that he is correct in separating them, and in support of his contention cites two other trematodes, *Opistorchis felineus* and *Opistorchis geminus*, which must likewise be recognised as separate species, though not presenting the slightest difference in their structural organisation. *O. felineus* is a European species, inhabiting the liver of certain beasts of prey, especially cats, and occasionally man. It has never been found in Egypt, neither in man nor in canine or feline animals, both wild and domestic, though purposely looked for. *O. geminus* is a common parasite in Egypt. It inhabits the liver of certain birds, amongst them the common Egyptian kite *(Milvus ægyptius)*, which never leaves the country, and must perforce acquire the parasite in it. It is quite reasonable, therefore, to infer that the *Avian opistorchis* indigenous in Egypt,

[1] Mesnil, F., Nicolle, M., and Remlinger, M. (January 22nd, 1908), "Recherche du protozaire de Wright dans 16 cas de bouton d'Alep." *Bull. Soc. Path. Exot.*, Vol. I., No. 1.

[2] *Loc. cit.*

[3] Sambon, L. (January 15th, 1908), "The Part played by Metazoan Parasites in Tropical Pathology." *Journal of Tropical Medicine and Hygiene*, Vol. XI.

could not be the same species affecting mammals and man in Europe, notwithstanding the total absence of structural differences. Looss's recognition of the two species, *C. sinensis* and *C. endemicus*, had already been disputed, but he (Dr. Sambon) was inclined to accept Looss's determinations, which he considered were founded on sound biological grounds. **Parasites—** *continued*

Amphistomum watsoni, a new trematode of man, found in the jejunum of a negro in German West Africa, and now assigned to the genus *Cladorchis*, is noticed, as are several new Tænia—notably *T. africana* found in German East Africa. Sambon also mentions his description of *Sparganum bæteri* from the same region, where it was found in the thigh of a native, and alludes to two new human *Linguatulidæ*, *L. serrata* and *Porocephalus armillatus*. He believes that five specimens of cylindrical linguatulid from the liver of African natives were really nymphal forms of *P. armillatus*, which is a parasite of the West African python. *P. armillatus* has never more than 22 rings, while *P. moniliformis* has from 28 to 30. He mentions various animals, including Ugandese monkeys, in which these nymphal forms have been found. The adult form occurs in African pythons and in the nose-horned viper, and hence its distribution should be coterminous with that of these ophidia, an area corresponding with that of the chimpanzee. This being so, it possibly occurs in a portion of the Bahr-El-Ghazal Province of the Anglo-Egyptian Sudan.

Sambon then proceeds to discuss bionomics and pathogeny, and a portion of this part of the paper may well be quoted :—

With regard to *A. lumbricoides*, the common lumbricoid worm of man, the general belief was that it reached the small intestines directly through the stomach, and, indeed, immature forms of this parasite had been passed by man. But he would point out that the larva of *A. lumbricoides*, like the larvæ of the above-mentioned species, was also provided with a perforating tooth which must obviously serve the same purpose, and that although we now believed that the development of this parasite occurred entirely within the one host, very competent observers, such as Leuckart, Brown and Von Linstow, had suggested that the larval stage might be spent in an intermediary host.

The *Gnathostomidæ* were again nematodes, the adult forms of which occurred in the stomach of vertebrates. In an earlier developmental stage they were usually found in cysts beneath the mucosa. One species, *Gnathostomum siamense*, had been found by Deuntzer in man in rounded nodules beneath the skin.

In the case of *Trichinella spiralis*, the adult forms seemed likewise to enter the lumen of the small intestines for the sole purpose of fertilisation ; the males usually died shortly after copulation. The whole period of growth was spent in a different part of the body, namely, in the connective tissue between the fibres of the striated muscles. The larvæ, extruded by the female worms into the interior of Lieberkuhn's glands, were carried into the circulating blood through the chyle vessels and thus reached the musculature.

Many nematodes entered their host in the egg or early larval stage, and although they invariably changed their anatomical habitat in the course of development, yet they did not leave their host, but completed within its body their entire life-cycle. Their ova never developed by their side within the same host, but must necessarily reach a fresh host after a shorter or longer period in the outer world. *T. spiralis* entered the intestine of a fresh host at the end of the larval period ; its young did not leave this host, but they migrated from the intestine to the musculature, and there developed until they reached a certain stage of growth beyond which they could not go unless their host were devoured by some other suitable animal. Thus, although all stages occurred within the same host, the life-history of the individual parasite was distributed between two hosts, which might belong to the same species (rats alone) or to different genera (rats and swine). In other cases, as in the majority of cestode infections, they found the larval or somatic stage in one host, the adult or intestinal stage in another host, usually belonging to widely sundered zoological groups. It was evident, therefore, that the life-history of each species of parasite, whether spent entirely in one host or distributed between two or more hosts, consisted of a larval, somatic stage, alternating with an adult stage located either within the intestines or in other parts (bile ducts, bronchial tubes, trachea, nasal fossæ) leading to the exterior. Occasionally the adult form inhabited the subcutis ; in such cases, as in *Dracunculus medinensis*, it perforated the skin at the time of parturition. Unfortunately, little or nothing was known with regard to the larval stages of most entozoa.

Time would not allow him to give more examples, or describe more fully the life-history of the parasites he had mentioned, but he hoped he had said enough to prove that an accurate and minute knowledge of the life-history of each species was indispensable to fully understand its pathogeny. Before accepting the theories of the chemist, who pounded worms in a mortar, he would like to hear the biologist. He believed that the migrations of the larval forms had not been taken sufficiently into account. Trichinosis was one of the few diseases in which the symptoms had been correctly ascribed to the agency of the larval forms. Now we should probably have to change our views with regard to the pathogeny of ankylostomiasis. The anæmic conditions of persons harbouring *Agchylostomum duodenale* could not be entirely ascribed to the direct abstraction of blood, since it had been practically demonstrated that the parasite was not a true blood-sucker. The anæmia of ankylostomiasis, like that of trichinosis might probably find its explanation, partly at least, in the actual migrations of the larvæ through the viscera and through the intestinal walls. The migrations of the comparatively large linguatulid nymphæ at the end of their encysted stage exemplified in a striking manner the nature of the process he wished to draw attention to. Writing on the pathogeny of *Porocephalus armillatus*, certain authors had expressed the opinion that this species is probably harmless to its hosts. He doubted whether such authors had ever set down the pen to handle the autopsy knife.

The paper is concluded by a few notes on the transmission of secondary infections by helminths.

Parasites—*continued*

Leiper,[1] in discussing the above, expressed his belief that the wanderings of immature helminths through the tissues of their host to their final habitat of sexual maturity or developmental arrest might well be the means of disseminating pathogenic agents within the body. As regards *Œsophagostomum brumpti*, he noted that an extensive diarrhœa and scouring in many of the lower animals was produced by species of this group, and he thought that in the Tropics the cysts and lesions of the epithelium associated with these worms might be overlooked.

A very suggestive paper is that by Ward[2] on the influence of parasitism on the host. After pointing out that during recent years there has been a tendency to exaggerate the unimportance of human parasites, he admits there are some of which it may fairly be said that even careful study has failed to show any manner in which they affect the host.

> Thus Looss (1894-3) records of a distome *(Heterophyes)* commonly found in the human alimentary canal among Egyptian labourers, that, although present in considerable numbers, most careful scrutiny fails to disclose any influence which it exerts upon the host. This is traceable to the fact that it neither burrows into, nor feeds upon, the mucous lining of the canal, but contents itself with taking its food from the partially digested contents of the intestine. Inasmuch as the organism is very small, this is evidently a negligible factor in the economy of the host; but even here, as I shall show later, there is the possibility that under some circumstances the organism may become a menace. Again, *Filaria loa*, the African eye-worm, lives for many years in the connective tissue of the human body, wandering from point to point, often not far below the skin. In the course of its migrations it does apparently no injury to the host, who is indeed unconscious of its presence until it happens to come into the connective tissue over the surface of the eye-ball. Here it appeals to the sense of sight, and from here it has most frequently been extracted. But in this case, again, there are swellings which appear from time to time on the surface of the body, and which are believed by some to be due in one way or other to this parasite. (*See* "Calabar Swellings," *page* 25).

Resting forms, such as bladder worms and young trichinæ, are also indifferent bodies, and the guinea worm exercises no influence on its host until the female appears at the surface. As factors determining the degree of influence exerted by the parasite on its host, he mentions the following :—

1. *Multiplication.*—Usually the single parasite leaves no effect. The multiplication is of course most serious when it takes place within the host and leads directly to a multiple infection. This is the case with some Nematodes, but in most metazoan parasites, including all Trematodes, Cestodes and some Nematodes this is not so, the eggs having to reach the outer world, and possibly an intermediate host, in order that development may proceed. Here the real danger lies in a multiple infection, through the increase in numbers which such a species often experiences in the intermediate host, or within a limited area in the outer world, so that by the taking in of a single external object a large number of parasites may be introduced.

2. *Size.*—In a general way the effects of a parasite are related to its mass as compared to that of the host, but from a special point of view this is absolutely untrue and the secondary effects of an individual species may be out of all proportion to its size.

3. *Site.*—This requires no comments. Contrast muscle fibre as a site with the brain or the eye.

The effects of a parasite on the host, Ward classes as (*a*) mechanical, (*b*) morphological, and (*c*) physiological. These may, of course, overlap.

(*a*) *Mechanical.*—Examples—Occlusion of a canal, say by a mass of round worms rolled into a ball. Arterial obstruction due to the young sclerostomes in the horse. The severe effects produced by *Schistosomum hæmatobium* on the capillaries.

Pressure effects, especially when the parasite is in a condition of active growth. There is also the mechanical effect produced by the movement of parasites, which may irritate and inflame the tissues. Migrating parasites have been known to cause death.

Again, the abrasion and destruction of surfaces and cells and the opening of abnormal communications may result in those secondary infections of a bacterial nature already mentioned. For example, *Ascaris* may penetrate the intestinal wall, and serious or fatal peritonitis result. Ward deals with the researches of Blanchard and Guiart, saying :—

> Evidently in producing ulcerations of the intestinal mucosa, parasites facilitate the absorption of toxins from the canal and permit the inoculation of this layer with pathogenic bacteria from the intestinal contents (Blanchard, 1904). They can thus be the agents of inoculation for numerous diseases. Guiart (1905), who defends this view most strongly, believes that intestinal parasites play an important rôle in the etiology of diseases

[1] Leiper, R. (February 1st, 1908), Report of Meeting of the Society of Tropical Medicine and Hygiene. *Journal of Tropical Medicine and Hygiene*, Vol. XI.

[2] Ward, H. B. (July 1st, 1907), "The Influence of Parasitism on the Host." *Proceedings of American Association for the Advancement of Science*, Vol. LVI.

Parasites— *continued*

of the intestine and liver, such as insects play in the etiology of blood infections. He advances evidence to support the view from the records of both human and comparative parasitology. While recognising fully that the infections are bacterial, he emphasises the necessity of some inoculating agent as, in a sense, the most important element, since pathogenic bacteria are generally present in the alimentary canal. No one can doubt, he maintains, that Eberth's bacillus is the agent of typhoid fever, but there is reason for regarding it as innocuous if the intestine is undamaged. In a population drinking contaminated water only a few persons in reality are infected. Any intestinal parasite capable of inflicting a wound may infect the host if the bacillus be present. The infecting agent may be an Ascarid, a hook worm, a fly larva; most commonly Guiart believes it to be the whip worm (*Trichuris*), which bores into the folds of the intestinal mucosa with its attenuated anterior end. This parasite Guiart (1901) calls the lancet of inoculation, and demonstrated its presence in eleven out of twelve typhoid cases in one group.

(*b*) *Morphological.*—It is difficult to find any definite explanation of the causes of the morphological changes which result from the influence of the parasite on its host. Ward suggests that the stimulation of growth by parasites may be due to a chemical stimulus, and that the stimulating substance is a poison, a proposition which brings us to

(*c*) *Physiological.*—1. The question of the absorption of nutriment takes first place in this connection. The parasite requires a certain amount of food matter to carry on its vital processes, and this is furnished, partially or fully digested, by the host animal. Some interesting details follow, but they cannot be given here.

2. Important effects are produced by increase in size of the parasite. Example: Interference with function produced by a large hydatid cyst of the liver.

3. Reflex nervous action. This, however, is pure hypothesis.

4. Retardation of development of host organism. Example: Parasitic castration (Giard).

5. Destruction of tissue of host. Example: The feeding of the liver fluke on liver tissue and the resulting growth of connective tissue. Much depends on the number of parasites concerned.

6. Inhibition of the coagulative power of the blood. Example: Agchylostomiasis. (Loeb and Smith).

7. Production of toxic material by the parasite. This may be waste, excretory matter, or it may be an actual toxic substance. Thus if an hydatid cyst rupture, serious results may ensue from the absorption of the toxic material liberated. A strong argument in favour of this toxic theory is the production of eosinophilia.

Production of anæmia as by *Dibothriocephalus latus*. The whole of this paper is well worthy of careful study and can be obtained in the form of a reprint.

A very similar article is that by Shipley and Fearnsides.[1] They take up the different groups of the metazoan parasites, and consider the effects produced by different species under each group. There is much of interest and numerous references, while special attention is devoted to the question of eosinophilia.

Weinberg[2] communicates an important paper on the parts played by helminths and their larvæ in the transport of pathogenic microbes. He believes that most helminths favour the incubation of the intestinal mucosa by microbes, and states that:—

The method of inoculation differs in different species of parasites. Thus certain nematodes, such as *Trichocephalus*, *Oxyuris*, *Sclerostoma*, *Physaloptera*, *Spiroptera*, which are capable of fixing themselves on the intestinal wall, inoculate directly with the microbes lying on the surface of their bodies. Others such as the *Ascarides*, though incapable of fixing themselves, favour infection by superficial gnawing of the mucous membrane and by causing small foci of congestion, which may develop into secondary centres of inflammation, and even of ulceration. Cestodes can also cause lesions of the mucosa. Although incapable of piercing it, their suckers give rise to foci of congestion, which may be inoculated with the microbes always to be found on these organs. A considerable infiltration of phagocytes follows, but under certain circumstances their action may not suffice to prevent inflammatory processes, and even ulceration.

Not only are helminths usually covered with microbes externally, but their intestinal contents may include a most extensive microbic flora, and they therefore constitute a serious danger to their hosts when they remain fixed for any length of time to the mucous membrane. Larval forms of these parasites penetrate the intestinal wall in large numbers, carrying microbes with them, and so set up submucous suppurations, aortitis and sub-peritoneal inflammatory nodules.

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[1] Shipley, A. E., and Fearnsides, E. G. (March 30th, 1906), "The Effects of Metazoan Parasites on their Hosts." *Journal of Economic Biology*, Vol. I. No. 2.

[2] Weinberg, M. (June 25th, 1907), "Du rôle des Helminthes, des larves d'Helminthes et des larves d'Insectes dans la transmission du Microbes pathogènes." *Ann. de l'Inst. Past.*, t. XXI.

Parasites— *continued*

The danger is, of course, directly proportional to the number of parasites harboured by the host, but even a single worm may bring about serious and even fatal consequences. A case where a single trichocephalus set up a fatal septicæmia from *coli* bacilli is instanced.

The author tried to set up typhoid fever in two monkeys harbouring large numbers of trichocephali. Both were fed several times with pure cultures of typhoid bacilli, and one died of typical typhoid.

The post mortem showed that in this case the penetration of the microbes was favoured, not by the trichocephali, but by a mass of tæniæ which obstructed the duodenum.

Lastly, he recalls to memory the polypi and adenomatous formations he has already recorded, arising on the points of fixation of helminths and intestinal larvæ.

There are some good illustrations showing polypoid and other conditions.

Shipley[1] has described the relation of entozoa to the mucous lining of the alimentary canal, and concludes that appendicitis might be caused by *Trichocephalus trichiurus*, but the general medical opinion as expressed by Manson is opposed to such a view.

Reference may here be made to a couple of general papers which are very useful to anyone studying helminthology. One by Ward[2] gives data for the determination of human entozoa, and although it is already somewhat out of date, the gaps can fairly easily be filled. The plates illustrating ova are very good. One table gives for each parasite in a lengthy list, name and organ infested, stage, *i.e.* larva or adult, type of parasitism, geographical distribution and recorded frequency as human parasite in normal habitat. A second table deals with the embryos of human parasites, detailing species, form, size in microns, surface, head, tail, sheath (presence or absence), and recorded presence or absence in blood, sputum, urine and fæces.

The other paper is by Stiles,[3] and furnishes an illustrated key to the cestode parasites of man.

Some work has been done on parasites found in animals in the Anglo-Egyptian Sudan, notably by Shipley,[4] while, in the collection made by Professor Werner of Vienna, Klaptocz[5] describes a new cestode in the guinea fowl, *Numida ptilorhyncha*, which bird seems to be the happy hunting ground of any number of blood and other parasites. (*See also* Dr. Leiper's paper in the Third Report.)

A new human intestinal parasite, *Physaloptera mordens*, has been found in Uganda, and is described by Leiper.[6] There is one other species of the same genus which infests man, *i.e. P. caucasica*, but the Uganda worm is known by its greater size, *Physaloptera mordens* measuring in the male specimens 29 millimetres in length and 2 millimetres in breadth and in the female 40 millimetres and 3 millimetres respectively; in the disposition of the papillæ of the male bursa and the contrast in the size of the spicules. In the female the situation of the vulva and the smallness of the egg are sufficient to separate the two species. Lastly, in the buccal armature an additional pair of teeth and two pairs of papillæ were noted.

Leiper[7] gives a partial description of a rare sclerostome found in the large intestine of a Nyasa native, and which proved to be *Triodontophorus diminutus*. This genus, *Triodontophorus*, was created by Looss in 1901 for certain blood-sucking sclerostomical forms found by him in Egyptian equines. The species in question was first described by Railliet and Henry in 1905. The females, which are carefully described by Leiper, occurred along with agchylostomes, the latter being in the small intestine.

Stephens[8] records two new human Cestodes and a new Linguatulid in man. One of the

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[1] Shipley, A. E. (March 28th, 1908), "The Relation of Entozoa to the Mucous Lining of the Alimentary Canal." Report of Meeting of Society of Tropical Medicine and Hygiene. *Lancet*, Vol. I.

[2] Ward, H. B. (1903), "Data for the Determination of Human Entozoa." Studies from Zool. Lab. Univ., Nebraska, No. 55.

[3] Stiles, C. W. (June, 1906), "Illustrated Key to the Cestode Parasite of Man." *Hyg. Lab. Bull.*, No. 25, Washington, U.S.A.

[4] Shipley, A. E. (1902), "On a Collection of Parasites from the Sudan." *Arch. de Parasit.*, t. VI., No. 4.

[5] Klaptocz, B. (1906), "Cestoden aus Numida Ptilorhyncha." *Lebt. Sitz. d. K. Akad. d. Wissen*, Wein.

[6] Leiper, R. (January 11th, 1908), "Physaloptera Mordens." Report of Meeting of Society of Tropical Medicine and Hygiene. *Lancet*, Vol. I.

[7] Leiper, R. T. (June 15th, 1908), "The Occurrence of a Rare Sclerostome of Man in Nyasaland." *Journal of Tropical Medicine and Hygiene.*

[8] Stephens, J. W. W. (February 29th, 1908), "Two new Human Cestodes and a new Linguatulid." *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. I., No. 4.

Cestodes, *Tænia bremneri*, is African, having been found in Northern Nigeria. It is figured in part and the measurements are given. Its great feature is the size of the proglottids, both as regards length and breadth. In the same journal will be found an illustrated contribution to the study of *Porocephalus moniliformis*, by Broden and Rodhain. **Parasites—** *continued*

Fleig and Lisbonne[1]* cite the work of Ghedini, who, in two human cases of hydatid cyst, proved the existence in the serum of specific anti-bodies, and that of Joest and Gherardini, who obtained negative results in the case of the echinococcus of animals. Fleig and Lisbonne found a specific precipitin (1) in the serum of a child, the subject of hydatid cyst of the liver (2) in that of animals inoculated with hydatid material, *i.e.* a maceration of the cyst membrane. The action takes place best at a temperature of 40° C. to 42° C.

After extirpation of the cyst there is a rapid disappearance of the precipitant power of the serum. Heating of the serum to 65° C. to 68° C. does not destroy the anti-body, but the reaction is hindered when the temperature of the hydatid fluid has been maintained at 61° C. for twenty minutes.

The liquid can be kept for at least two months and utilised for the sero-diagnosis of individuals suspected of harbouring hydatid cysts. The results obtained so far, both from a positive and negative aspect, have been excellent, and have been controlled surgically.

One may conclude with a note[2] on oil of filmaron recommended for *T. saginata*, *T. solium* and *Dibothriocephalus latus*, 1 part to 9 parts of castor oil. The oil (children 90 grains, adults 150 to 180 grains), in capsules, may be given in two doses, with half an hour interval, by itself, followed by the castor oil in requisite dose one or two hours later, after the rectum has been washed out by a water and glycerin enema.

Filmaron, it may be added, is an amorphous acid extracted by Böhm from male fern.

Paratyphoid Fever. Hewlett[3] has a paper on the subject, and mentions that as regards the Widal reaction the blood of the paratyphoid patient either does not agglutinate the typhoid bacillus or agglutinates it only in low dilution, *e.g.* 1 in 30 to 40, while it agglutinates the paratyphoid bacilli in far higher dilution, *e.g.* 1 in 100 or 200, or even higher (in one case 1 in 8000). The paper, which deals with bacteriology and symptomatology, is useful, but our knowlege has considerably increased since it was written.

Perhaps the best recent article in English is that by Birt,[4] who states that paratyphoid fever is not a well-defined entity. It is (he says) impossible to find a diagnostic point by which it may be separated from enteric. The complications are seldom serious, and the mortality seems to be about 2 per cent. There are no definite post mortem appearances by which paratyphoid fever can be recognised. He deals with the A and B varieties of the bacillus, and gives a useful table of cultural differences. Paratyphoid A resembles the *B. typhosus* more closely than paratyphoid B, but infections due to the former in man are rare. The paratyphoid group is, however, widely diffused in Nature, being found in the intestinal canal of healthy animals, etc. He concludes (1) that paratyphoid infections cannot be distinguished clinically from enteric, than which they are less common ; (2) a negative serum reaction with the enteric bacillus or a positive reaction with a paratyphoid bacillus is not sufficient to justify a diagnosis of paratyphoid fever ; (3) in every febrile case blood cultures should be made at once for diagnostic, prognostic and therapeutic purposes.

Very much the same conclusions are reached by Poggenpohl,[5] who points out that the agglutination reactions cannot be relied upon, and that clinically the recognition of paratyphoid affections is of no importance. He hopes that it may yet be possible to convert *B. coli* into *B. typhosus*, a procedure which, if accomplished, would doubtless do much to clear up what is at present obscure regarding the precise significance and relations of the various paratyphoid bacilli.

Fox[6]* has a good paper on the subject, albeit perhaps a little out of date. He says :—

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[1] Fleig, C., and Lisbonne (June 29th, 1907). *C. R. Soc. Biol.*, t. LXII.

[2] "Filmaron for Tapeworm." *Journal of Tropical Medicine and Hygiene*, 15th February, 1908, Vol. XI.

[3] Hewlett, R. T. (January, 1904), "Paratyphoid Fever." *Practitioner*, Vol. LXXII., No. 1.

[4] Birt, C. (August, 1907), "Typhoid and Paratyphoid Fevers." *Journal of the Royal Army Medical Corps*, Vol. IX.

[5] Poggenpohl, S. M. (August 29th, 1907), "Zur Diagnose und zum Klinischen Verlauf des Paratyphus." *Zeit. für Hyg. und Infekts.*, Vol. LVII., No. 2.

[6] Fox, H. (July, 1905), "The Nature of Paratyphoid Fever." *Med. Chron.* Quoted in *Indian Medical Gazette*, October, 1905, Vol. XL.

* Article not consulted in the original.

Paratyphoid Fever— *continued*

1. Paratyphoid fever differs from typhoid fever in *(a)* a shorter invasion stage and rise of temperature, *(b)* shorter or absent period of continued fever, and *(c)* marked diurnal remissions of temperature, much deeper than enteric and without periodicity. An absence of the Widal is suggestive if it persist in reasonably high dilutions.

2. The duration is, on the whole, shorter than typhoid, and in the cases where type "B" was adjudged the etiological rôle this fact is more striking than in the type "A" cases.

3. The general findings of the type "A" cases are nearer to typhoid than type "B," the latter presenting a picture more like septicæmia.

4. The complications of type "B" infections are more numerous, more purulent, and the course is more fulminating in these cases.

5. The causal germs belong to the intermediates of the typhocolon series, the type "A" being nearer to the bacillus of Eberth and Gaffky, while type "B" approaches the meat-poisoning group.

6. The clinical evidences of the respective organisms just named agree with their general properties and relations to infections in this order, ranging from the sub-acute typhoid to the hyper-acute meat-poisoning.

7. Anti-typhoid serum will clump the paratyphoid "B" at the same time as the *B. typhosus*, sometimes even in higher dilutions; so that a positive reaction of a patient's serum to both *B. typhosus* and paratyphosus "B," even if the latter be in higher dilutions, will not permit a diagnosis. On the other hand, only twelve times in 94 cases of fever did the serum react with the type "A" paratyphoid, so that a positive reaction with type "A" and not with the bacillus of Eberth, may be taken as nearly a proof of the existence of an "A" paratyphoid infection. (But *vide infra*).

8. That there must be some other factor responsible for co-agglutinations than an increased value of the agglutinins normally present in the blood seems probable.

Castellani[1] has studied the condition in Ceylon and has noted the occurrence of mixed infection. He concludes that Ceylon must be included among the countries where paratyphoid fever is endemic, both types of the disease ("A" and "B") being encountered; that the disease cannot be distinguished from typhoid, though it generally runs a milder course; that in one case of "A" infection, intestinal ulcers were found, and that cases of mixed infection are apparently not rare.

Rogers[2] deals with the disease in India, where he believes several varieties probably exist. In favour of this view he cites Castellani's Ceylon cases already mentioned under "Bacteriology" (*page* 12).

MacNaught[3] has also described new forms of paratyphoid bacillus, which he isolated from the blood of cases occurring at Wynberg in Cape Colony. He gives tables showing the cultural characteristics of the bacilli isolated.

Henry[4]* states that the pathology differs widely from that of typhoid fever, the autopsy findings being largely those of a septicæmia. Where ulceration of the intestine has been found, he maintains, in contra-distinction to later authors, that the ulcers are of a dysenteric type and do not affect Peyer's patches. He places the mortality at about 6 per cent. Vagedes[5]* records a case of paratyphoid poisoning attributed to infected ducks' eggs, which were used in the making of a cake; and Conradi[6]* has reported the simultaneous presence of *B. typhosus* and *B. paratyphosus* in a water which had fallen under suspicion.

The blood of a patient suffering from fever was sent me from Wad Medani on the Blue Nile. It gave negative agglutination reactions in dilutions of 1 in 20, 1 in 40, and 1 in 100, a positive result with Paratyphoid "A" in 1 in 20, but not in 1 in 40, or 1 in 100; but a definite and complete reaction in all three dilutions with Paratyphoid "B."

In the absence of blood culture, one cannot be certain, but it is very probable that this was a case of paratyphoid fever which in all probability exists in the Sudan, though, like enteric fever, it cannot be at all common.

Piroplasmosis. This subject, so far as the Sudan is concerned, will be found considered in a special paper. Here one proposes to deal with some general papers of interest and then consider articles dealing respectively with the equine, bovine and canine piroplasmoses.

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[1] Castellani, A. (February 2nd, 1907), "Paratyphoid in the Tropics: Cases of Mixed Infection." *Lancet*, Vol. I.

[2] Rogers, L. (London, 1908), "Fevers in the Tropics."

[3] MacNaught, J. G. (February, 1908), "A Note on Two Cases of Paratyphoid Fever, in which a New Variety of Paratyphoid Bacillus was found in the Blood." *Journal of the Royal Army Medical Corps*, Vol. X.

[4] Henry, J. A. (April 15th, 1905). *Amer. Med.*

[5] Vagedes, K., "Paratyphus-bazillen bei einer Mehlspeisevergiftung." *Klin. Jahrb.*, Bd. XIV.

[6] Conradi, H. (1907), "Ein gleichzeitiger Befund von Typhus und Paratyphus-bazillen in Wasser." *Klin. Jahrb.*, Bd. XVII., fax. 2.

* Article not consulted in the original.

Piroplasmosis—*continued*

Of special interest is the work of Koch[1] on the development of the parasite in the tick. His observations were made on *P. bigeminum* and *P. parvum* of bovines. The process only occurs in adult females which have gorged themselves with blood. The changes begin after 12 to 20 hours in both species when the parasites leave the erythrocytes and lie in heaps. Some develop spear-like processes and become club-shaped. These spears resemble pseudopodia. Two chromatin granules are seen at the thick end of the parasite. The spears become less numerous and the parasites more rounded. They grow and acquire a membrane. Amœboid bodies are developed which break up into rounded parasites each with a chromatin mass. Similar forms were seen in the eggs of the tick. Kleine[2] found somewhat similar changes in *Babesia canis* kept in defibrinated blood mixed with normal saline.

Koch's observations have been confirmed and extended by Christophers,[3] who worked with *Piroplasma canis* in India. As regards the developmental cycle in the tick, he concludes:—

1. In *R. sanguineus* there are two means by which infection is transmitted.

(*a*) Hereditarily through the egg. A method shown both by experimental infection of dogs and observation of the parasite in the tick.

(*b*) Stage to stage infection. Not yet proved by experimental infection, but practically certain from observations upon the parasite.

2. In both methods of infection the parasite goes through the same cycle of development, becoming in turn a club-shaped body and then a zygote which breaks up into "sporoblasts," and these again into "sporozoits."

3. In hereditary infection club-shaped bodies originating each from a single parasite penetrate the ova either in the ovary or in their passage down the oviduct, and in the yelk become zygotes. In the larva the zygotes have broken up into sporoblasts which are found disseminated in the tissues, and in the nymphs the sporozoits have accumulated in large numbers in the salivary glands.

4. In infection from nymph to adult the club-shaped bodies, after being formed in the gut of the nymph, penetrate cells of the embryonic tissue which will eventually form the adult, and embedded in the cells of this they become zygotes. The sporozoits derived from these zygotes may find themselves, without any action on their part, in salivary cells or they may be situated elsewhere, in which case they probably reach the salivary cells by their own movements, possibly aided by the circulation of fluids in the tick.

The details of development strongly suggest a cycle of a sexual nature, and, if this be the case, the sexual cycle of piroplasma has many points in common with the sexual cycle of the malarial parasites and proteosoma. This has been already pre-supposed by the nomenclature employed which it seems reasonable to use until further research either confirms or shows it to be untenable.

The greatest difference between the development and that of the malarial parasite occurs in the peculiar dissemination of the sporoblasts and sporozoits, and the fact that the ookinete (?) comes to rest not in the gut wall but in the tissues. That the malarial zygote has no true wall of its own has been already supposed by Grassi, and in this the zygote of piroplasma would bear it a resemblance. The separation of sporoblasts by the growth of the embryonic tissue, and possibly by the movements of the sporoblasts themselves and the infiltrating action of piroplasma, have, so far as I know, no parallel in malaria or other of the pathogenic protozoa. The sporozoits, except that they have not the filiform shape of those of malarial parasites, seem to correspond exactly to these and their eventual location in the salivary acini is exactly parallel to conditions in malaria. Though it is clear much has still to be done in following out details, there can be no question that in the main the mystery surrounding the passage of piroplasma through the tick has been solved.

The paper is very well illustrated and the bibliography is very full. In a later article[4] he says:—

Stated briefly, the hereditary cycle of *Piroplasma* is as follows:—

A parasite in the gut of the adult tick enlarges and becomes a motile, club-shaped body, which then leaves the gut and penetrates an ovum, becoming in the substance of this a zygote. The zygote increases in size and breaks up into sporoblasts, which are found disseminated in the tissues of the larva. In the glands of the nymphs immature sporozoits have collected. In the glands of the adult are found mature sporozoits.

Development in the nymph to adult infection is identical. Club-shaped bodies are formed in the gut. They leave this, penetrate the tissues, embedding themselves in cells and becoming zygotes. Owing to the growth of the embryonic tissue, the sporoblasts into which the zygote breaks up tend to become disseminated among the cells. Many of these invaded cells later form the salivary gland—a structure which occupies a large portion of the body of the infected tick. The sporozoits thus find themselves *in situ*. Many parasites, however, invade embryonic cells which do not eventually become salivary tissue, and the sporozoits then probably reach the gland by their own movements.

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[1] Koch, R. (1906), "Beiträge zür Entwicklungsgeschichte der Piroplasmen." *Zeits. f. Hyg. und Infekts.* Bd. LIV., No. 1.

[2] Kleine, F. K. (1906), "Kultivierungs versuch der Hunde Piroplasmen." *Ibid.*

[3] Christophers, S. R. (1907), "Piroplasma Canis and its Life Cycle in the Tick." *Scientific Memoirs of the Government of India*, No. 29.

[4] Christophers, S. R. (November 9th, 1907), "Development of Piroplasma Canis in the Tick." *British Medical Journal*, Vol. II.

Piroplasmosis— *continued*

Passing now to the consideration of *Equine piroplasmosis*, which has been found by Olver to occur in the Sudan, into which country, however, it appears to have been recently imported, one may note a paper by Bowhill,[1] who describes the South African form attacking the horse, mule and donkey. He describes large and small spherical forms, large and small pyriform parasites, large and small rod-like bodies, the rosette form which is a division stage in reproduction and resembles a Maltese cross, a St. Andrew's cross or the Manx coat of arms, and the flagellate forms which have a pear-shaped head and a long flagellum ending in a bulbous protuberance. He also found spherical or ovoid, extra-corpuscular forms in blood preserved aseptically in a flask with sterile citrate of potash solution at room temperature. The symptoms are given, there being acute and chronic forms. There is intense fever at the onset, and in the acute type, lachrymation, disinclination to move, and a stumbling gait. Later on there is paresis of the hind limbs, and coma followed by death in a few hours. The other chief symptoms are anorexia (though the animal may be voracious), icterus, anæmia and weak and irregular pulse. There may be diarrhœa, or constipation with dark, foul and slime-coloured fæces. Occasionally there is hæmoglobinuria. Bowhill states that many cases recover without any special treatment, while some are benefited by small doses of sodium bicarbonate. Hutcheon recommends belladonna and ammonium chloride. It would seem that South African veldt horses are more or less immune, the immunity depending apparently on the animal being reared in an infected area. A page is devoted to secondary or terminal infections, and Theiler's observation quoted to the effect that—

> It is exceedingly rare to find that only the piroplasma is present in a horse suffering from, or dying of, biliary fever. In nearly every case I found a bacterium (a cocco-bacillus showing bi-polar staining) which was present sometimes in the blood, and always in the spleen.

Theiler[2] studied the transmission of the disease by ticks, and concludes that *Rhipicephalus decoloratus* is not a host of *Piroplasma equi*, while *Rhipicephalus evertsi* transmitted the parasite in its adult stage after feeding as larva and nymph on a sick horse. It is, therefore, a host. There is not yet sufficient proof to show whether the disease is transmitted through the egg of a tick.

He has also drawn attention[3] to the risk of inducing the disease in horses utilised for hyper-immunisation. He found that the greatest risk of causing piroplasmosis by infusion is in animals which are hyper-immunised for the first time. There is still a certain amount of risk in subsequent infusion, probably due to the first virus horse not being immune against piroplasmosis. It is noteworthy that a horse which has undergone an inoculation of piroplasmosis, and shown *Piroplasma equi* during the reaction, may still contract the disease from hyper-immunisation. This contingency, therefore, has to be expected whenever piroplasmosis immune animals are utilised for hyper-immunisation purposes.

Roger[4]* has described what he calls equine petechial piroplasmosis in Algeria. He recognises benign, hæmoglobinuric and grave forms. The special symptom seems to be the presence of petechiæ on the conjunctiva and *membrana nictitans*. In the grave form, where there is a "typhoid" condition, the pituitary membrane is also dotted over with petechial spots.

The parasites found outside the erythrocytes were rounded in form, those within were either spherical or pyriform and resembled bacilli. Roger differentiates it from other forms described, by the fact that (1) at first the membranes are not icteric, and (2) all the cases have shown petechiæ on the conjunctiva and *membrana nictitans*, a symptom which only occurs in severe forms of equine piroplasmosis.

Jolliffe[5] has reported on the disease as encountered in India. He queries the conveyance of the disease by ticks—well-groomed horses being affected, and is inclined to think that blood-sucking flies may be at fault. As regards treatment, he thinks that quinine, with or without salicylate of soda, gives the best results. He also gives the differential

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[1] Bowhill, T. (January, 1905), "Equine Piroplasmosis or Biliary Fever." *Journal of Hygiene*, Vol. V., No. 1.

[2] Theiler, A. (December 31st, 1906), "Transmission of Equine Piroplasmosis by Ticks in South Africa." *Journal of Comparative Pathology and Therapeutics*, Vol. XIX.

[3] Theiler, A. (1905-6), "Report of Government Veterinary Bacteriologist, Transvaal Dept. of Agriculture."

[4] Roger, J. (December 31st, 1906), "A Form of Equine Piroplasmosis seen in Algeria." Quoted in *Journal of Comparative Pathology and Therapeutics*, Vol. XIX.

[5] Jolliffe, C. H. H. (February, 1907), "Some Remarks on Equine Biliary Fever in India." *Journal of Tropical Veterinary Science*, Vol. II., No. 1.

* Article not consulted in the original.

diagnosis from simple jaundice. The latter may be recognised by (1) its rapidly favourable course, (2) the absence of any marked constitutional derangement and the trivial character or non-existence of pyrexia, and (3) by the presence, usually, of bile in the urine. **Piroplasmosis—***continue*

In the same journal is a translation of a paper by Pricolo, who shows that the so-called typhoid fever of the horse is really piroplasmosis.

Williams[1] also deals with the Indian disease. He lays great stress on the peculiar conditions of the conjunctival mucous membrane, which he believes to be characteristic. It is reddish-brown in colour, with a tinge of yellow, and a few bright red petechial spots are scattered over the *membrana nictitans*. These petechiæ gradually increase in size and alter in colour considerably during the first few days of the disease; they become more of the nature of blotches, and may coalesce to form comparatively large patches, the colour changing through various shades of red to a deep claret, and this latter appearance is reached about the fourth or fifth days.

He likewise advocates quinine in large doses (2 drachms) at the onset, decreasing the quantity after the first few days; and he is inclined to think that in India, blood-sucking flies and not ticks are the vectors.

Bovine Piroplasmosis. This subject as it affects the Sudan, receives mention in a special paper wherein will be found references to three forms, which have been discovered in Sudanese cattle. One of these is *P. mutans*, first described by Theiler in South Africa. In a paper[2] on this species, he gives a list of the piroplasmata of cattle known up to date (end of 1906).

Type.—Piroplasma bigeminum. P. bovis (Babes), found in the European hæmoglobinuria of cattle, *P. bigeminum* (Smith and Kilborne) of Texas fever.

Type.—Piroplasma parvum. A. Inoculable piroplasmosis. Tropical piroplasmosis of Trans-Caucasia. *Piroplasma annulatum* (Dschunkowsky). *Piroplasma mutans*, n. sp., of South Africa. B. Non-inoculable piroplasmosis. *P. parvum* (Theiler) of East Coast fever. Piroplasma of the North African Disease (Bitter and Duchoux).

In this paper, proofs are given to show the duality of *P. bigeminum* and *P. mutans*, and in a later article[3] further proof is advanced, and in addition it is shown that the blue tick, which is the carrier of *P. bigeminum*, does not transmit *P. mutans*.

In another paper[4] he describes and figures the curious "marginal points" found in the erythrocytes, the nature of which is still unknown. They are situated on the periphery of the corpuscle, are round or oval, and exclusively take the chromatin stain. Theiler states that they are to be regarded as a sequel of ordinary redwater, but I have reason to believe he has since altered his opinion on this point (Olver). I have seen these bodies in cases of *P. mutans* in the Sudan. Proceeding, Theiler says:—

In the Transvaal, at the present time, there are three different piroplasmoses known to exist in cattle: (1) one due to *Piroplasma bigeminum*, and commonly called "redwater"; (2) one due to *Piroplasma parvum*, and known by the name of East Coast fever; and (3) one due to *Piroplasma mutans*, for which a specific term does not exist, but it probably ranges under the name of "gall sickness."

The first and third of these diseases are inoculable—*Piroplasma bigeminum* and *Piroplasma mutans*. Immune cattle contain the parasites in their blood. In both diseases calves easily recover from the infection, whereas, under natural conditions, adult cattle suffer more severely. Cattle born in the Transvaal usually acquire immunity against both diseases, hence the imported ones suffer principally in this respect.

Piroplasma bigeminum causes a disease after a short incubation time, and, being deadly for imported cattle, destroys a large number before *Piroplasma mutans* has time to develop, hence cases due to this latter disease are comparatively rare. It is also probable that this second disease is constantly mistaken for redwater, and this will continue unless microscopical examinations of blood are made.

Piroplasma mutans has a practical importance in connection with East Coast fever.

Piroplasma parvum may easily be, and has constantly been, mistaken at various times for *Piroplasma mutans*. The presence of small piroplasmata in rare numbers is, therefore, not always indicative of East Coast fever.

For diagnostical purposes in such cases, examinations of blood must be repeated. In East Coast fever, the piroplasms will usually rapidly increase in numbers, whereas *Piroplasma mutans* increases slowly and is never present in large numbers.

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[1] Williams, A. J. (March, 1907), "Indian Equine Piroplasmosis." *Journal of Comparative Pathology and Therapeutics*, Vol. XX.

[2] Theiler, A. (December 31st, 1906), "Piroplasma Mutans (n. spec.) of South African Cattle." *Journal of Comparative Pathology and Therapeutics*, Vol. XIX.

[3] Theiler, A. (March 30th, 1907), "Further Notes on Piroplasma Mutans, etc." *Journal of Comparative Pathology and Therapeutics*, Vol. XX.

[4] Theiler A. (1905-6), "Report of Government Veterinary Bacteriologist," Transvaal Dept. of Agriculture.

Bovine Piroplasmosis— *continued*

It sometimes happens, however, that animals in an infected area die before *Piroplasma parvum* has developed to any extent, and in such cases the diagnosis must remain doubtful. Robertson has found that East Coast fever may run its course with a total absence of *P. parvum* in the peripheral blood, or perhaps only with the presence of a very small number of these parasites. In such cases a post mortem examination is the only way of enabling one to form a correct diagnosis. Since, however, *P. parvum* does not always produce the typical lesions (infarcts in the liver, etc.), even this procedure may be useless.

It will be remembered that, in the case of East Coast fever, transmission by the bite of the progeny of ticks fed on sick animals, always failed to infect; but infection taken in by the larvæ was transmitted by the nymph, and that taken in by the nymph was transmitted by the adult (Lounsbury and Theiler).

Quite recently Fülleborn[1]* has found cross-shaped divisional forms in *P. bovis*, so that these can no longer be regarded as characteristic of *P. parvum* and allied species.

A condition likely to be of interest to veterinarians in the Sudan, is that ascribed by Kowalewski[2] to an atypical form of piroplasmosis, which seems really to be the latter disease complicated with rinderpest, as is evidenced by the post mortem findings. In endeavouring to differentiate the disease from others, the following points should be borne in mind:—

1. The epithelium surrounding the erosions and small swellings on the under lip is firmer, and cannot so easily be rubbed off as in rinderpest.

2. In piroplasmosis the small swellings are firmer, white in colour, isolated, and are not covered with a caseous material as in rinderpest.

3. In piroplasmosis cases are observed where no swellings whatever exist, but only numerous little erosions.

4. The enlargement and softening of the spleen, which only occurs exceptionally in rinderpest.

5. Very characteristic ochre-like coloration of the liver.

6. Hæmorrhagic processes in the kidneys; and

7. In a few cases, blood-stained urine.

Of very considerable importance, if it be confirmed, is the announcement by Miyajima[3] that he has succeeded in cultivating a bovine piroplasm found in Japanese cattle, and apparently identical with *P. parvum*, because *in vitro* the parasites took on the trypanosome form. The simple method employed is as follows:—

The blood containing intracellular parasites is drawn from the jugular vein and then quickly defibrinated under strict precautions so as to avoid bacterial contamination; it is then directly mixed with ordinary nutrient bouillon, in proportions varying from one-fifth to one-tenth, and placed aseptically in sterile test-tubes, which thereafter are maintained at a temperature of 20° C. to 30° C. The development of the parasites in a successful culture takes place in the following manner: On the first day no motile form is seen; on the second, there can be observed a certain number of peculiar cells, which occupy the upper layer of sedimented corpuscles and which macroscopically appear as a series of whitish dots. Very few motile forms resembling typical trypanosomata are visible in these cells on the third day after incubation, but thereafter the trypanosomata multiply vigorously and reach the maximum number between the tenth and fourteenth days.

In a culture kept at room temperature, the trypanosomata remain motile until forty-five days later; at this time most of them have undergone degeneration and globular cells with irregular granulations result. In a culture preserved at a lower temperature, ranging from 10° C. to 20° C., the organism on the contrary remains alive until three months after the maximum number has been reached. It is noteworthy that subcultures are also readily obtained by inoculating from the original strain into a new blood bouillon, as in the case of *Trypanosoma lewisi*.

The most important factor in securing the multiplication of the parasites essentially consists in great precautions in avoiding the slightest contamination with bacteria, as is the case with other cultures of protozoa.

With reference to preventive measures, Captain Olver kindly furnished me with the following particulars regarding the *rationale* and carrying out of Stockmann's method, which, associated with the paying of appropriate indemnities, has proved very successful in South Africa:—

1. East Coast Fever is conveyed by ticks only, and no other animals except cattle appear to be susceptible.

2. Cattle which have recovered from an attack do not harbour the parasite, and consequently are incapable of acting as permanent centres of infection.

3. Infected ticks clean themselves and are incapable of infecting susceptible cattle afterwards, by feeding on non-susceptible animals.

Consequently, if a farm is kept free of all cattle for a sufficiently long period to allow all existing ticks to go through a complete life-cycle the disease dies out and will not re-appear unless re-introduced from outside. Fortunately ticks do not travel far unless carried.

In South Africa, where the system was successfully applied in practice, the period was arbitrarily fixed at 14 months, but it is probable that even less would be sufficient.

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[1] Fülleborn (1908). *Arch. für Schiffs. und Tropen. Hyg.*, Bd. XII.

[2] Kowalewski, I. M. (June, 1907), "Clinical and Anatomical Appearances of the Atypical Form of Piroplasmosis." Quoted in *Journal of Comparative Pathology and Therapeutics*, Vol. XX.

[3] Miyajima, M. (May, 1907), "On the Cultivation of a Bovine Piroplasma." *Philippine Journal of Science*, Vol. II., B. Med. Sc.

* Article not consulted in the original.

Theiler[1] reports efforts in the case of East Coast fever to produce a serum which has no hæmolytic action on the blood of sick animals, and acts as a preventive on healthy ones. These did not meet with success, but were found to have a bearing as regards rinderpest prevention. One cannot give further details here. **Bovine Piroplasmosis—** *continued*

Bugge[2]* mentions fairly good results obtained in the protective inoculation of cattle against redwater by the injection of sterile, defibrinated blood from calves which have recovered from the disease at least fifty days. These inoculations should be carried out from four to six weeks before the animals are sent to grass. The precautions to be taken are described.

The occurrence of piroplasmosis in the goats of Central Africa is signalised by Panse.[3]* The forms seen recalled *P. parvum* of cattle. Piroplasmata have also been found in sheep and in deer.

Canine Piroplasmosis. A great deal of experimental work on this form has recently accumulated. Thus we have the important papers of Nuttall and Graham-Smith,[4] who have studied the parasite as it is found in the peripheral blood, both in fresh and stained films. Their work, which is profusely illustrated, led them to the following conclusions:—

1. Nearly all forms of piroplasma show one densely staining mass of chromatin—the nucleus; many also show a second punctiform dense mass generally situated near the nucleus—the blepharoplast; and a considerable number show a third loose mass which has not been previously observed. These masses may occupy various positions, or assume various shapes, the significance of which we have not yet been able to determine.

2. Many intracorpuscular forms in stained preparations show both pseudopodia and flagella-like processes, and many of the free forms possess distinct flagella.

3. Round forms, apparently in a degenerating condition, are common in liver and spleen smears. In these situations free masses of chromatin also occur, probably derived from degenerated parasites.

4. Many of the appearances seen in stained preparations are extremely deceptive, and deductions made from them are frequently not confirmed by the study of living forms.

5. Various bodies occur in normal dog's blood, which may readily be mistaken for piroplasmata.

6. *Piroplasma canis* has a truly intracorpuscular and an extracorpuscular stage. In the former condition, round, amœboid and pyriform bodies occur, all of which are to some extent motile, and in the latter, long and pyriform, frequently flagellated free swimming bodies. Amœboid extracorpuscular forms are never seen.

7. Within the peripheral blood a definite cycle of development occurs. Free pyriform bodies invade the corpuscles, become round and later amœboid. The amœboid bodies, according to their size, either again form intracorpuscular pyriform bodies or divide and form two or more pyriform bodies. The pyriform bodies leave the corpuscles, and in doing so, rupture them and enter other corpuscles.

A free pyriform parasite enters a normal red blood corpuscle and rapidly assumes a rounded form. It then enlarges and passes through an actively amœboid stage, at the end of which it again becomes rounded. After a short period of quiescence in this condition it protrudes two symmetrical processes, which rapidly grow and become pear-shaped. The protoplasm of the parasite flows into these processes, and its body consequently gradually diminishes until it is represented by a minute rounded mass to which the pyriform processes are attached. Eventually this also disappears, and finally two mature pyriform parasites are left, which are joined together for a time by a thin strand of protoplasm. After a variable time these parasites are liberated by the rupture of the corpuscle, and swim away to enter fresh corpuscles and repeat the process.

Occasionally a single, rounded intracorpuscular parasite, by the protrusion of several processes such as have just been described, gives rise to four or more mature parasites, or a single parasite divides into two small rounded parasites, each of which produces two pyriform parasites.

Under experimental conditions all parasites, which are liberated by the rupture of the corpuscles containing them before they have reached the mature pyriform parasites which fail quickly to enter fresh corpuscles, disintegrate and die.

We have never observed any forms which could be regarded as gametes.

Christophers'[5] monograph is not only an excellent dissertation on *Piroplasma canis*, but contains a review of the whole subject of piroplasmosis and a consideration of the ticks which attack dogs. One or two points dealt with in his review may be mentioned:—

1. The blood of recovered animals is infective, even though parasites may not be demonstrable under the microscope.

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[1] Theiler, A. (July, 1907), "Experiments with Serum against East Coast Fever." *Journal of Tropical Veterinary Science*, Vol. II., No. 3.

[2] Bugge (March, 1908), "Protective Inoculation against Redwater in Cattle." Quoted in *Journal of Comparative Pathology and Therapeutics*, Vol. XXI.

[3] Panse (1908). *Arch. für Schiffs. und Tropen. Hyg.*, Bd. XII.

[4] Nuttall, G. H. F., and Graham-Smith, G. S. (July, 1905, October, 1906, April, 1907), "Canine Piroplasmosis." *Journal of Hygiene*, Vols. V., VI., VII.

[5] *Loc. cit.*

* Article not consulted in the original.

Canine Piroplasmosis— *continued*

2. The injection of blood containing piroplasmata into animals, of a species other than the normal host of the parasite, has so far in no recorded case been followed by success.

3. Lounsbury's work showed that in South Africa piroplasmosis of the dog is conveyed by the tick *Hæmaphysalis leachi;* not by the larvæ from infected mother ticks, but only by ticks which, reared from eggs laid by infested mother ticks, had passed through two metamorphoses and had reached the adult stage. This is, of course, different from what obtains in *P. bigeminum* infection, where the transmission is conveyed by the larva of the second generation, and quite different from the method already mentioned for *P. parvum* infection (*page* 151.)

Christophers has shown that for *P. canis* still another process is available, for, in the case of transmission by *R. sanguineus*, nymphs of the second generation may be infective. This is a matter of interest in the Sudan, where "Yellows" of dogs is very common and where *R. sanguineus* is the tick concerned.

An acute and chronic form of the disease is seen in dogs. In the former there is complete anorexia, and, save in very young dogs, where a high temperature is absent, there is marked fever. Anæmia, jaundice, weakness and paresis of the hind legs complete the clinical picture.

The chronic form is associated with intense anæmia, but rarely with icterus or hæmoglobinuria. Fever is not a prominent sign, but there is emaciation, anorexia and a scurfy skin.

As regards immunity, Nocard and Motas noticed not only a high degree of immunity resulting from previous infection lasting many months, but also showed that the blood of immunised dogs has a marked bactericidal effect upon the parasites, mixture *in vitro* with four or five times its volume of serum from an immunised dog rendering virulent blood ineffective. The serum of immunised dogs has, however, but slight effect in preventing or modifying an attack.

The pathological changes are described and, under the heading "Morphology," the following forms of parasite receive mention: amœboid, pear-shaped, large, early, infection forms, round refractile type, ring, vacuolated, flagella-like processes, post mortem small globular forms, and free forms frequently occurring in groups.

Most have found the incubation period by natural infection to lie between ten and twenty-one days, but in Christophers' experiments it was very often only four days.

One cannot consider the paper in greater detail, save for a few notes as regards dog ticks. After mentioning these and their geographical distribution, Christophers says:—

R. sanguineus is probably the species having the widest distribution, and would in itself serve as a transmitter in Southern Europe, North Africa and much of the East. For South Africa there is a special dog tick, *H. leachi*. In Europe and more temperate regions, the various species of *Ixodes* seem to act as carriers. In Japan the only dog tick noted is an *Ixodes*, and an *Ixodes* is found on dogs in Australia. In Central America several species of Amblyomma seem to be associated with the dog. A chapter on the general life-history of ticks, and especially of *R. sanguineus*, will be found useful. The experimental infection work has already been noted (*page* 152).

Kinoschita's work,[1]* performed under the auspices of Schaudinn, differentiates 1. Round, amœboid, schizogonic forms. 2. Pear-shaped, sexually distinct forms. The former proliferate by a process of budding, the latter, both male and female, by longitudinal binary division. It is thought that the male and female gametocytes conjugate in the tick, but no proof is advanced. Post mortem the parasites are as numerous in the capillaries of the skin as in those of the internal organs. It would appear that this work requires confirmation.

The latest work on the life-cycle of *P. canis* emanates from Breinl and Hindle,[2] of Liverpool, who, in the study of the parasite, used the fixing and staining methods introduced by Breinl for trypanosomes, *i.e.* the fixing of *wet* films by strong Flemming's solution, and staining with safranine and methylene blue, or by a modified Heidenhain's method, a dilute solution of Bordeaux red being used as a counter stain. The authors describe early and late forms, and, in connection with the latter, specially mention the occurrence of unequal divisions of the parasite. They also deal with the flagellated forms, and trace the development of the large bi-flagellate forms from the normal intracellular parasite. This brief notice of a useful paper, illustrated by two coloured plates, must suffice.

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[1] Kinoschita, K. (1907). *Arch. f. Protistenkunde*, p. 294.

[2] Breinl, A., and Hindle, E. (July 1st, 1907), "Contributions to the Morphology and Life History of *Piroplasma Canis*." *Annals of Tropical Medicine and Parasitology*. Series T. M., Vol. II. No. 3.

* Article not consulted in the original.

Canine Piroplasmosis—*continued*

Hutcheon[1] has a good paper on biliary fever in dogs, and quotes Lounsbury as follows :—

Like all other Ixodinæ, both the cattle tick and the dog tick pass through three active life stages, viz., the larval, nymphal and adult. Preparatory to the transformation from one stage to the next, the feeding of the tick is suspended; thus, after an interval of variable duration, the old skin is ruptured, the tick crawls from it, and very soon seeks to renew its attack. The cattle tick normally stays on one animal throughout the several changes. It remains attached to the skin by its rostrum. After it suspends feeding, both as a larva and as a nymph, and hence, on the completion of the moult which follows, it has only to re-attach itself to the animal by its new mouth part, an act which it performs almost at once. The dog tick, on the other hand, perhaps because it infests an animal more energetic than an ox in locating and destroying skin parasites, has the habit of invariably loosening its hold and seeking shelter in the ground as soon as it ceases feeding before a moult, and, in consequence, it has the trouble of finding a host three times in its life instead of once, as with the cattle tick, and more often than otherwise a different dog is found each time.

But a more troublesome peculiarity than the change of a host at each moulting, was the fact, experimentally proved, that although the infective parasite is passed from the infected mother tick through the egg to the larval tick hatched from it, yet, although this young tick evidently harbours the infective parasite, through its larval and nymphal stages, it cannot communicate the parasite to a susceptible host until it arrives at its mature or adult stage.

He mentions that Robertson failed to confirm the results obtained by Nocard and Motas as regards prolonged immunity (*see page* 154), but agrees with their observations on phagocytosis. Speaking of preventive and curative remedies, he says :—

There can be little doubt that if a safe and effective method of preventive inoculation could be discovered, one that could be applied say every six months in order to maintain the immunity, that would be the most satisfactory method, and I am not without hope that such a means may be discovered.

He recommends calomel and quinine as early as possible in the disease.

Wetzl[2]* records a case of recovery under careful feeding with eggs, sugar, milk and treatment with ferrum peptonatum cum arsenic. Gonder[3]* treated ten dogs with atoxyl in varying doses, but was unable to observe any effect either on the incubation period or the course of the disease.

Plague. The more the Sudan is brought into contact with the outside world the greater the risk of invasion by plague. Plague is endemic in Uganda and on the eastern shore of the Red Sea, while of late years Egypt has been constantly re-infected. Hence the whole question of plague is one of very considerable importance, more especially since the establishment of traffic on the Port-Sudan-Atbara railway.

Naturally papers on preventive methods are those likely to be most useful, but certain others will be considered as well. Klein,[4] in his recent book, when discussing the analysis of plague materials, alludes to the great importance of using at once plate cultures in preference to tube cultures of solid or fluid media, and, later, has some useful notes on microbes simulating *B. pestis*. Amongst these are *B. proteus vulgaris*, *B. coli*, *B. bristolense*, an organism found in dead rats on ship-board and showing marked bi-polar staining, *B. myxoides* and *B. muris*. He indicates how mistakes may be avoided, and lays stress on the necessity for examining any organism obtained from a suspicious case in hanging drop preparation. This in itself will exclude many bacteria, for *P. pestis* is, of course, non-motile.

MacConkey[5] has a paper on the *B. pseudo-tuberculosis rodentium* (Pfeiffer) which, both morphologically and culturally, resembles *B. pestis* more than any other known organism. He found that, by inoculations of this bacillus, it was possible to immunise guinea pigs and rats against *B. pestis*, the immunity sometimes lasting for a period of six months.

In dealing with protective inoculation, Klein describes the preparation of his own new plague prophylactic, made from the organs of animals dead of plague, and compares it with Haffkine's fluid, saying :—

When it is borne in mind (1) that this dried prophylactic does not require more than about ten to twelve days for its preparation—Haffkine's requires four to six weeks; (2) that a large amount can be prepared of uniform strength; (3) that its efficacy is easily standardised by injection into the rat; (4) that, being dry and sterile, it can be preserved without any antiseptic and unaltered for any length of time; and (5) that the protection afforded by its injection into the rat is of considerable duration, certainly many weeks; and last, but not least, that the cost of preparation is incomparably smaller; the superiority of this *organ-prophylactic* to Haffkine's prophylactic must be obvious.

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[1] Hutcheon, D. (November, 1907), "Biliary Fever in Dogs. Malignant Jaundice or Canine Piroplasmosis." *Journal of Tropical Veterinary Science*, Vol. II., No. 4.

[2] Wetzl, T. (February, 1908), "On Piroplasmosis in Dogs." Quoted in *Journal of Tropical Veterinary Science*, Vol. III, No. 1.

[3] Gonder, R. (1907). *Arb. a. d. K. Gesundheitsamte*, Bd. XXVII, fasc. 2.

[4] Klein, E. (London, 1906), "The Bacteriology and Etiology of Oriental Plague."

[5] MacConkey, A. T. (June, 1908), "On the Relationship between Bacillus Pseudo-tuberculosis Rodentium (Pfeiffer) and Bacillus Pestis." *Journal of Hygiene.*

* Article not consulted in the original.

Plague— *continued*

This is all very well, but Klein's prophylactic had, at the time this was written, only been tested on animals, and it has yet to stand the severe trials from which the older prophylactic has triumphantly emerged. This leads us to speak of Haffkine's views on plague and his preventive method, and, where much is confused and contradictory, one is glad to find a paper at once so clear and free from dubiety. Haffkine[1] first of all enumerates the measures which have been suggested for stamping out the plague or preventing its importation. The first category of measures comprises :—

1. Discovery and notification of persons attacked with the disease.
2. Isolation of the attacked.
3. Certain precautions with regard to the disposal of the dead.
4. Segregation of those who have come in contact with the sick or dead.
5. Institution of cordons round infected areas.
6. A less drastic and less thorough plan than the last mentioned, viz., placing in quarantine arrivals from infected places, detaining the sick and suspected, and letting the rest free after a time of observation; or
7. A still less rigorous measure, which is merely to examine travellers, isolate the sick and suspected, and let the others free under a system of surveillance.

He deals with these in detail and concludes that all the evidence goes to show that (*a*) plague is what has been termed in a general sense a disease of locality ; (*b*) it is contracted principally at night ; and (*c*) that the part which man plays as a direct agent in its propagation is a more or less subordinate one. Hence the futility of the above measures, which are always difficult, often impracticable, and can achieve little of value.

The second list of measures is directed against places and fomites *within an infected area*, and comprises destruction or disinfection of houses, furniture, clothing, bedding, carriages, goods, warehouses, grain and other stores, garbage, drains and streets. *Outside the infected area* the measures consist in the refusal to admit carts trains and ships with goods from infected places; or in the refusal to admit only certain goods; or in mere inspection of trains, carts and ships, and some procedure by which these, and the goods they convey, as well as the belongings of travellers, are sought to be rendered harmless.

All these measures are intended for the avoidance or destruction of plague germs which may possibly exist in the objects concerned.

He notes, however, that the expense and difficulty of such operations are enormous and that the ascertained facts regarding plague bacilli do not appear to justify their adoption.

The third category of measures relates to the lower animals, and includes :—

1. Destruction or keeping away of rats by poisoning, trapping, tar and sulphuric acid mixture, or through the agency of the domestic cat.
2. Improvements in towns and villages, with a view of reducing or keeping out the rat population, viz., structural alterations of dwellings, warehouses and grain stores, demolition of insanitary buildings, introduction or improvement of conservancy arrangements, prompt disposal of garbage, periodical inspection of stores, paving and draining of streets, and certain other measures.
3. Destruction and dispersion of fleas by petroleum or other insecticides.
4. Fumigation of houses as a temporary protection against rats and fleas.
5. Obligation on ships from infected regions to anchor away from the shore; or
6. Provision of mechanical arrangements for preventing the landing of rats along mooring cables and gangways; and
7. Fumigation of ships arriving with plague patients or plague rats on board.

The measures have, therefore, for their object, and, I believe, rightly so, the rat and the flea, described by Rothschild under the name of *Pulex cheopis;* but epizootics of plague break out also among squirrels, tarbagans, guinea pigs, mice, monkeys, kangaroos in Australia, and some other animals, which contribute to keeping the disease alive.

Haffkine, however, cites the experiences in rat destruction at Sydney, New South Wales and in Japan, where the results of well-organised campaigns against these rodents were far from being encouraging. He goes on to say that though the measures against rats, either extermination or by change in the construction of cities and villages, are a most important item in an anti-plague campaign, the question whether any noticeable impression can be made on the epidemic by these measures within the length of a generation, or even in a longer period, is a matter of great uncertainty. Even the destruction of rats on ships alone, if imposed as a general measure, would cause a dislocation of traffic and an outcry formidable to face. The result is that every day, plague is imported, though fortunately it need not spread, into one part or other of the marine countries of the world.

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[1] Haffkine, W. M. (February, 1908), "On the Present Methods of Combating the Plague." *Journal of the Royal Institute of Public Health*, Vol. XVI.

Plague—*continued*

He then considers the plan of abandoning an infected locality for shorter or longer periods. This, which may be termed a *second stage* in a campaign against plague, while at times useful and salutary, is often impracticable and may inflict great hardship and suffering. To whatever extent it is feasible, to that extent the effect of it is beneficial.

By a process of exclusion he thus arrives at what he calls the *ultimate method* of combating bubonic plague in the areas in which it becomes endemic, viz., that of conferring on the population immunity from the disease by means of an artificial treatment, *i.e.* preventive inoculation.

He tabulates the salient facts established as regards this method during the last ten years of Indian experience as follow :—

1. That in a native of that country, who is more susceptible to the disease than Africans, Europeans and some other races, the inoculation now in force in India reduces the liability to attack to less than one-third of what it is in a non-inoculated Indian.

2. That in the one-third of cases which still occur the recovery rate is at least double that in the non-inoculated attacked, the ultimate result being a reduction of the plague mortality by some 85 per cent. of what it is in non-inoculated Indians.

3. That in an inoculated European, an attack of plague, if it subsequently occur, has so far always ended in recovery.

4. That the inoculation is applicable to persons already infected and incubating the plague, and prevents the appearance of symptoms, or else mitigates the attack, a fact which disclosed a basis for the bacterio-therapeutic treatment of disease.

5. That in natives of India, the degree of immunity conferred by this inoculation, though gradually vanishing, seems to last during several outbreaks of plague; and that

6. In Europeans, the effect has not yet been seen to disappear in the space of time, since 1897, that this inoculation has been under study.

Haffkine's able advocacy of his method is confirmed by Simpson,[1] who first of all cites the conclusions arrived at by Haffkine in connection with the outbreak at Byculla jail. These were :—

(1) That one injection of 3 c.c., of the prophylactic was sufficient to protect during an existing epidemic; (2) that inoculation was powerless to arrest the disease in those in whom the symptoms have already appeared or develop in a few hours after inoculation; (3) that the inoculation mitigated or aborted the disease in those who were in the incubation stage, and had been infected three or four days previously; (4) that the prophylactic, unlike the vaccines for cholera, rabies, anthrax, or small-pox, exercised its protective effect in less than twenty-four hours, acting in this respect with a rapidity which was only known in antitoxic sera.

He then proceeds to give tables on the working of Haffkine's system both on a small and on a large scale which leave no manner of doubt as to its value and efficiency.

As regards the duration of immunity, it would seem that the effect of the inoculations lasts for four or five years, though gradually it diminishes.

At this stage it may be well to give the instructions issued in the Punjab Plague Order[2]* for performing inoculation against plague :—

Hot vaseline is used for sterilising syringes, needles and instruments. Kapadia's lamp, which consists of a spirit lamp, with a pot for vaseline and a thermometer attached, is recommended for sterilising purposes. When the vaseline reaches a temperature of 90° C., the moisture in the syringe is rinsed out with vaseline; and when a temperature of 160° C. is reached the syringe is sterilised by being filled and emptied three times. The neck of the bottle containing the prophylactic is sterilised by means of a flame, or by dipping the bottle into the hot vaseline, and when the neck cracks the tip is knocked off by sterile forceps.

The site of inoculation should be prepared as follows :—The patient's arm should be thoroughly washed with soap and water, then dried; then thoroughly washed with cyllin and water (1 in 40); and then a small square of lint soaked in cyllin lotion (1 to 20) should be applied over the exact place where the puncture will be made.

Before each inoculation the needle must be dipped into vaseline at 160° C.

Should any blood ooze from the puncture on the withdrawal of the needle, a piece of lint soaked in cyllin lotion (1 to 20) should be applied.

Whenever the syringe has to be laid on the inoculating table it should be placed on one piece of lint and covered with another piece, both pieces of lint being thoroughly soaked in 1 to 20 cyllin lotion.

At the conclusion of each day's operations the operator should himself rinse out with cyllin lotion (1 to 20) all syringes that have been in use, and should sterilise all his needles and instruments in vaseline heated to 160° C., thus also protecting them from rust.

A lotion of cyllin of 1 to 20 strength is also prescribed for the bowls containing the spare needles and forceps.

Substitution of cyllin as a disinfectant in place of perchloride of mercury in these manipulations is worthy of note.

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[1] Simpson, W. J. (August 1st, 1907), "Croonian Lectures on Plague, Lecture III." *Journal of Tropical Medicine and Hygiene*, Vol. X.

[2] "Plague Inoculation Manipulations." Quoted in *Journal of Tropical Medicine and Hygiene*, March 16th, 1908, Vol. XI.

* Article not consulted in the original.

Plague— *continued*

Sandwith[1] has a good account of plague in Egypt, but one need not quote the description of clinical symptoms. The method of procedure for obtaining plague material for bacteriological diagnosis recommended by the Egyptian Sanitary Department may, however, be detailed.

The syringe together with the needle should be sterilised in boiling water, immediately before being used.

The syringe should be placed in a recipient containing cold water in such a quantity as to allow the syringe to be entirely covered with water. The water will then be heated to boiling point and kept boiling for fifteen minutes. Should the piston of the syringe not fit well, the screw at the extremity of the piston rod should be well tightened. It is necessary to ascertain that the syringe works properly before it is sterilised.

Before the puncture is made the skin to be punctured should be first cleaned by rubbing it thoroughly with soap and water and a piece of clean cotton wool. Then it should be rubbed with cotton wool wet with corrosive sublimate (1 in 1000), and finally with cotton wool wet with pure alcohol.

The liquid withdrawn into the syringe must be deposited on the surface of the agar by pressing the piston. The operation should be carried out with great care in order to avoid contaminating the agar by other bacteria.

The cotton wool plug of the tube of agar should never be touched with the hand, except on the end which is outside the tube.

The cotton wool plug should under no circumstances be placed on the table or on any other place.

The tube before being put into the wooden box should be well wrapped round with cotton wool in order to avoid its breaking during transport. These boxes should always be posted as ordinary letters and not as parcels.

Immediately after use, the syringe should be sterilised again in the following manner:—

Fill and refill the syringe several times with water withdrawn from the sterilising vessel: this must be continued until no trace of blood or pus remain in the syringe.

During the emptying of the syringe the needle opening must always be kept beneath the water, to avoid any small drops of infected matter being by chance spread about.

The syringe and needle are then placed in the sterilising vessel and the water heated to boiling point, and kept boiling for fifteen minutes.

The syringe is to be used only for puncturing suspicious cases of plague.

Typhus fever, according to Sandwith, is the disease most liable to be confounded with plague in Egypt. He gives at some length the instructions for disinfection issued by the Egyptian Sanitary Department, whose success in controlling the spread of plague is universally recognised. Whether this is due to these disinfection measures is, however, I venture to think, a point which requires to be determined in the light of the work of the Indian Plague Commission.

Sandwith also deals with rat destruction and quarantine, giving the regulations in force at Tor, on the Red Sea.

A review in the *Lancet* of September 8th, 1906, of the special plague pamphlet issued by the above Department may be quoted in full, as the conditions in the Sudan closely resemble those obtaining in Egypt.

The Director-General of the Public Health Department begins by reminding all officers that the same care must be exercised in disinfection and the general management of isolated cases as in dealing with distinct outbreaks. It is this individual care so far exercised in Egypt, which has chiefly contributed to the success of anti-plague measures. Directly a living case of plague is discovered a police guard should be placed on the house, and the disinfector and the head man of the quarter must be sent for. The probable source of infection should be ascertained and a list made of all contacts, which includes the inhabitants of the house and the nearest relatives. The patient must be removed to the infectious hospital or to a special building or temporary hut which can be utilised as one. The contacts must be examined every day for ten days, and disinfected. If a disinfecting station exists in the town, all clothes and soft goods must be removed there; if not, the clothes must be disinfected with the premises. As soon as possible after the patient's arrival at the hospital, some serum should be withdrawn from his bubo (or lung in the case of pneumonic plague) with all aseptic precautions; the drop or two of serum ejected on to agar must be sent to Cairo or Alexandria without delay. When the patient is already dead, the specimen for bacteriological examination must be procured in the place where the body is lying, corrosive sublimate (1 in 1000) is given for washing the corpse, and the shroud must be soaked in the same solution; the bier should be lined with zinc or tin and disinfected after use. It is very seldom found necessary to destroy infected clothes, but they should be soaked in sublimate solution for 20 minutes and afterwards thoroughly dried in the sunlight. In all cases of pneumonic plague the contacts must be kept strictly isolated for ten days. It has been found that when concealment of cases is practised in the village a secret gratuity of one dollar for each report of a fresh case of plague has an excellent effect. In the case of a foreign subject, neither disinfection nor removal to hospital can take place without previous consultation with the Consul concerned. When the disinfection of a house is finished, all rat holes must be thoroughly opened up, a small quantity of carbolic solution (1 in 10) poured into them, some broken glass placed so as to plug the bottom of the rat run, and held in position by mortar, and the hole mortared up flush with the wall. All dead rats and mice found by the workmen are put into a special paraffin tin and covered with carbolic acid. Rat poisoning is best carried out with phosphorus paste concealed in tomatoes. I may here mention that phosphorus and arsenic are the poisons, together with traps, found most useful in Japan. The number of rats killed in Tokyo since 1900 averages more than 800,000 a year, and it is calculated that these dead rats, laid side by side, would extend for a distance of over

[1] Sandwith, F. M. (London, 1905), "The Medical Diseases of Egypt," Part I.

Plague— *continued*

75 miles. Yet, according to Kitasato, "we can hardly notice any considerable decrease in the number of these animals in Tokyo." Mosques in Egypt must be treated with respect by the disinfecting gangs, but the manager of the mosque is given sublimate solution, so that the mosque servants may carry out disinfection. The most convenient form of hospital hut for rapid building is found to be ten metres long by six metres wide, divided by a partition for the two sexes.

We now pass to a consideration of the work of the Indian Plague Commission.[1] Considering their experiments and conclusions *seriatim*, one notes that they found that close contact of plague-infected animals with healthy animals, if fleas are excluded, does not give rise to an epizootic among the latter; that close contact of young, even when suckled by plague-infected mothers, did not give the disease to the former; that if the fleas are present, then the epizootic once started, spreads from animal to animal, the rate of progress being in direct proportion to the season of the year and the number of fleas present; that an epizootic may start without direct contact of healthy animal and infected animal; that the rat flea can convey plague from rat to rat; that infection can take place without any contact with contaminated soil; and that aerial infection can be excluded.

Another series of experiments carried out in plague houses in Bombay showed:—

1. Guinea pigs allowed to run free in plague houses in many instances attracted a large number of fleas, which fleas were mostly rat fleas. A certain percentage (29) of these animals contracted plague and died from the disease. The position of the bubo, in the great majority of these cases, was cervical.

2. If a plague house had been previously disinfected by the ordinary means of disinfection, fleas were still caught in large numbers on guinea pigs set free in them. Further, a considerable number (29 per cent.) of these animals died of plague. The bubo, in the great majority of these cases, was again in the cervical region.

3. Fleas transferred from plague-infected rats found dead or dying in houses were able to transmit plague to healthy animals in flea-proof cages in the laboratory. The bubo, in all cases, was in the cervical region.

4. Fleas transferred from guinea pigs and other animals which had been placed for a few hours in plague houses were able to transmit the disease to fresh animals when fed on these in flea-proof cages in the laboratory. The situation of the bubo in these animals was, in the great majority of cases, in the cervical region.

5. Animals were placed in plague houses in pairs, both protected from soil and contact infection and both equally exposed to aerial infection, but one protected from fleas by means of a fine metallic curtain and the other not so protected. None of the protected animals contracted plague, while several of the unprotected animals died of the disease. The position of the bubo, in every instance, was in the cervical region.

6. Animals were placed in plague houses in pairs, both protected from soil and contact infection and both equally exposed to aerial infection, but one surrounded with a layer of "tangle-foot" and the other surrounded with a layer of sand. The following observations were made:—

(*a*) Many fleas were caught on the tangle-foot, a certain proportion of which were found on dissection to contain in their stomachs abundant bacilli microscopically identical with plague bacilli. Out of 85 human fleas dissected, only one contained these bacilli, while out of 77 rat fleas 23 were found thus infected.

(*b*) The animals surrounded with tangle-foot in no instance developed plague, while several (24 per cent.) of the non-protected animals died of the disease.

Work on rat plague is concluded by the following summary:—

It has been shown that plague rats, like human cases, may be divided into two classes, according as to whether or not a bubo is present. The bubo, if present, is the most important diagnostic sign of plague.

Of other characteristic appearances, those occurring in the liver of plague-infected rats have been described in detail, since they are of primary importance from the point of view of diagnosis. Hæmorrhages in various parts of the body are commonly met with, and an abundant clear pleural effusion constitutes, when present, a noteworthy sign of plague in the rat.

Analyses of the results of microscopical examination of 1200 plague rats are set forth in the form of tables. It is apparent from these that the bubo gives the best chance of recognising plague bacilli in large numbers. Not only so, but the value of the bubo as an aid in the microscopical diagnosis of plague is increased by the presence in at least 50 per cent. of those examined of the characteristic involution forms.

Reference has been made to the occurrence of plague-like bacilli or of plague-like diseases in rats. We can only reiterate the statement that in Bombay no difficulty of this kind has been experienced.

The relative value for diagnosis of the macroscopical and microscopical methods of diagnosis has been discussed. The results of tests carried out for the purpose of comparison, make it manifest that the naked eye is markedly superior to the microscopical method as an aid in diagnosis, and, as the result of our experience, we are prepared to make a diagnosis of

[1] "Transference of Plague from Rat to Rat." *Journal of Hygiene*, September, 1906, Vol. VI.

"Further Observations on the Transmission of Plague by Fleas, etc.," July, 1907. *Ibid.*, Vol. VII.

"The Epidemic and its Relation to the Epizootics," December, 1907. *Ibid.*, Vol. VII.

Plague— *continued*

plague on the strength of the macroscopical appearances alone, even though the other results of cutaneous inoculation and culture are negative, and the animal shows marked signs of putrefaction.

The value of the method of cutaneous inoculation of guinea pigs has been examined; it would appear to fail only in about 2 per cent. of fresh, and about 10 per cent. of putrid, rats.

The bacilli found in naturally infected rats are fully virulent, 62 per cent. of the inoculated animals die of acute plague in five days or less. A well-illustrated paper on the pathological histology of the spleen and liver in spontaneous rat-plague, which follows the above, will repay perusal. Rats were fed with infected material, and it was found that:—

1. It is possible to infect wild rats of Bombay with plague by feeding them with the viscera of dead plague rats, 21·4 per cent. being susceptible to this method of infection. Bombay rats show a greater immunity to infection by feeding than rats of the same species, which have not been subjected to a plague epizootic.

A series of experiments were also done with *Mus rattus* caught in the Punjab. Of these rats 67·8 per cent. were susceptible. In this series a considerably larger dose of infected material was given.

We have infected a large number (38 per cent.) of wild Bombay rats by feeding them on the whole carcases of their plague-infected comrades. No difference as regards the post-mortem appearances or the distribution of the primary bubo was found between rats infected in this way and rats infected by feeding on soft viscera.

2. The general pathological lesions found in all rats infected by feeding are, in the main, the same as those found in rats naturally infected. There are, however, two striking differences:—

(*a*) The distribution of the primary bubo is different. The common site in naturally infected plague rats is in the neck, no mesenteric bubo having been seen out of 5000 post mortems. In the case of fed rats the common site is the mesentery.

(*b*) In the case of naturally infected rats the stomach and intestines show no marked pathological change. In the case of fed rats well marked pathological lesions are found in the intestines.

3. It would appear that in nature intestinal infection rarely or never takes place, and that in consequence rats do not become infected by eating the carcases of their comrades.

4. A large series of rats were fed on the urine of plague cases. None of these contracted the disease.

Most important of all, perhaps, are the extended observations on the transmission of plague by fleas and the fate of the plague bacillus in the body of the rat flea (*P. cheopis*). Thus:—

1. The average capacity of a rat flea's stomach is approximately 0·5 c.c. On this basis a flea imbibing the blood of a plague rat showing a good septicæmia might take as many as 5000 germs into its stomach.

2. Multiplication of the plague bacillus takes place in the stomach of the rat flea.

3. The approximate proportion of fleas in the stomach of which multiplication of plague bacilli takes place has been determined, and it has been shown that this proportion varies with the season of the year, being six times greater in the epidemic season than in the non-epidemic season.

4. Plague bacilli are present in the rectum and fæces of fleas taken from plague rats, and such fæces are infective to guinea pigs both by cutaneous and by subcutaneous inoculation.

5. On rare occasions plague bacilli have been found in the œsophagus, but never in any other region of the body, such as the body cavity or salivary glands.

6. During the plague season fleas might remain infective for 15 days after imbibing infective blood, but during the non-epidemic season no animal was infective after the 7th day.

7. A single rat flea may transmit the disease.

8. Both male and female rat fleas can transmit the infection.

9. Experimenting with cat fleas (*P. felis*) and human fleas (*P. irritans*), 27 experiments with the former were unsuccessful, and out of 37 experiments with the latter three successes were obtained. Two experiments were made with *C. fasciatus*; both were successful. Multiplication of the plague bacillus takes place in the stomach of the human flea.

10. The plague bacillus has never been seen in the body cavity or in the salivary glands of infected fleas.

Evidence has been obtained to show that the bite of a healthy flea affords a sufficient avenue for infection by septicæmic blood if it be spread upon the bitten part. No evidence has been obtained in favour of infection by contaminated mouth parts or regurgitation from the stomach, but the possibility of infection by such means cannot be excluded.

There is also a paper on the differentiation of the rat flea from other fleas.

As regards the epizootic amongst rats, the following points were determined:—

1. *Mus decumanus* and *Mus rattus* are equally susceptible to plague.

2. The incidence of plague is twice as great on the *decumanus* population as on the *rattus* population.

3. *Mus decumanus* is the species which is chiefly responsible for the diffusion of plague amongst the rats throughout Bombay City.

4. The *decumanus* epizootic precedes the *rattus* epizootic by a mean interval of about ten days.

5. The *rattus* epizootic is directly attributable to the *decumanus* epizootic.

6. Plague persists in the rats in Bombay City during the off season. This persistence is due chiefly to *M. decumanus*.

M. decumanus, it is noted, harbours twice as many fleas as *M. rattus*.

Plague—*continued*

A study of the epidemic and its relation to the epizootic showed :—

With regard to the incidence of plague on different classes of the population, we may note that little difference, if any, exists in the liability to infection of males and females; that there is a varying incidence on persons of different age-periods, the greatest incidence being on persons between 11–20 years of age; and that of the different races in Bombay, Hindus and Mahomedans suffer most severely from the disease.

We may summarise our conclusions regarding the inter-relations of the epidemic and the epizootics as follows:—

1. The time-relation of the epidemic and the *rattus* epizootic is explicable on the view that the rat flea is the transmitting agent of the infection from *M. rattus* to man.

2. From the point of view of place-infection, there is an intimate relation between the epidemic and the *rattus* epizootic.

3. There is a definite quantitative relation between the incidence of human and of rat plague.

4. The epidemic is directly attributable to the *rattus* epizootic, and since this epizootic is in its turn directly attributable to the *decumanus* epizootic, the epidemic is indirectly attributable to the latter epizootic.

While the last conclusion expresses the broad relations of the epidemic and the epizootic, it must be added that:—

5. Infection is occasionally transferred directly from *M. decumanus* to man, *i.e.* without the intervention of *M. rattus*.

Another important list of conclusions is as follow :—

1. The question of the alleged spread of infection by direct contact with a suffering case has been discussed. Our observations in a plague hospital, and with material obtained from this hospital, lead us to conclude that such a mode of spread does not exist. Support is given to this view by a consideration of the influence of imported cases on the spread of the epidemic and by an investigation of the relative frequency of single and multiple cases in houses and buildings. We have, further, referred to our experience that a rat epizootic is alone sufficient to account for a widespread dissemination of infection throughout a locality. A review of the whole of the evidence on this point brings us to the conclusion that contact plays no part in the spread of the epidemic.

2. In discussing this question, the question of the infectivity of houses, evidence has been brought forward which points to the rat flea being the transmitting agent of infection from rat to man. Further, reasons have been given for the view that plague does not persist in a locality apart from infection amongst the rats.

3. From the arguments brought forward in the discussion of the two previous questions we conclude that the epidemic is wholly dependent upon the epizootics.

4. It has been shown that infection may be transported to a distance by means of rat fleas in clothing or merchandise, and that such infection, when imported into a hitherto uninfected locality, may give rise to an epizootic in the rats.

5. Our observations lead us to conclude, that plague in domestic animals in Bombay either does not occur, or occurs so seldom that it cannot be said to possess any significance from an epidemiological standpoint.

A study of the insanitary conditions prevailing in Bombay led to the conclusion that these exert no direct influence on the spread of epidemic plague, although ill-constructed buildings, by affording shelter to rats, undoubtedly indirectly facilitate its diffusion.

Hunter,[1] of Hong Kong, in a paper written some time ago, but none the less interesting, drew attention to the prevalence of gastro-intestinal lesions in plague, and to the fact that diarrhœa and vomiting often constitute the earliest symptoms.

It is suggested that the alimentary tract is the avenue by which the plague bacillus enters the human body. It must be confessed that the evidence put forward is very suggestive, though later work has not confirmed this view. The presence of skin eruptions in plague is noted ; papules, vesicles and pustules being frequently seen. Hunter's work tended rather to discredit the flea theory and to support the view that insects, such as cockroaches, and possibly bugs and spiders, may carry the bacilli to food, which thus becomes the vehicle of infection, and which, of course, may also become infected in other ways. This theory seems to have been somewhat lost sight of, and the enquiries of the Indian Commission serve, in some measure, to discredit it, but the last word has not been said on plague. Should it revive, one would like to suggest that ants be added to the list of insects likely to be harmful. In the Sudan at least, of all the insects one has studied, these seem by far the most likely to bring about infection of food and drink as mentioned under the heading " Cholera " (*page* 30). It is hoped to carry out some investigations on this point.

[1] Hunter, W. (January 2nd, 1905), " A Research into Epidemic and Epizootic Plague." *Journal of Tropical Medicine and Hygiene*, Vol. VIII.

Plague— *continued*

Lamb,[1] a member of the Commission, in replying to certain criticisms by Hossack, has some interesting notes. He deals specially with the multiplication of plague bacilli in the flea's stomach, where everything favours their increase. The fæces of a flea are full of plague bacilli, and while it is sucking blood it is constantly squirting out fæces from the anus. Thus a mass of bacilli are deposited near or on skin abrasions, a condition most favourable for infection. He concludes by saying that the facts proved are (*a*) that bubonic plague in man is entirely dependent on the disease in the rat; and (*b*) that infection is conveyed from rat to rat, and from rat to man solely by means of the rat flea.

França[2]* has also drawn attention to the skin lesions in plague, making special mention of the carbuncles which may be present, and which differ from those found in anthrax, the œdema not being so pronounced and the surrounding ring of vesicles often absent. Pemphigus, he notes, is rare.

The Indian Medical Gazette for July, 1906, constitutes a special plague number, and it is remarkable how many divergent views are expressed. Those of Browning Smith appear to be most in line with the findings of the Indian Plague Commission. A few of his conclusions may be quoted:—

1. The flea can retain the plague bacillus alive and virulent from seven to eight days (Ziroglia). 2. Man is, in the large majority of instances, infected through the skin, though the breach of continuity is too small to be detected. 3. The rat flea, when deprived of its host, will attack man and animals other than the rat. 4. The rat flea has been found on plague-stricken man and animals other than the rat.

In India the four fleas commonly associated with man and rats are—1. The cat flea, *P. felis* v. *serraticeps*, small and dark; (2) The human flea, *P. irritans*, large and somewhat light-coloured; (3) The black rat flea of the black rat *(Mus rattus)*, *P. cheopis*, small, light-coloured; (4) The brown rat flea of the brown rat *(Mus decumanus)*, *Ceratophyllus fasciatus*.

A few of our Sudanese fleas have been identified, mostly by Rothschild. One knows that *P. cheopis* is common, and hence Rothschild's[3]* description may be given:—

This species is larger than *P. nubicus*, the palpus being shorter than the rostrum and not reaching to the end of the coxæ. In the male, sternites, three to seven inclusive, bear four bristles; while those of the female have five. The hind femur bears, in addition to the lateral series of hairs, two subventral bristles before the apex. The first segment of the mid-tarsus less than two-thirds the length of the second, while that of the hind tarsus is about three-quarters as long again as the second segment. The long apical bristles of the second segment of the hind tarsus reach to the middle of the fifth segment in the ♂, and not quite so far in the ♀. The fourth segment is as in *P. nubicus*. The eighth sternite bears two long bristles before the end on each side, and numerous short ones besides. The anterior process of the clasper of the male is compressed, being asymmetrical in shape. The upper, or anterior, edge is convex, bearing along this edge a number of rather long bristles. The second process of the clasper is slender, with a few short hairs at its end. The ninth sternite gradually widens towards the apex. The plate of the penis is curved upwards and pointed at the end.

It has been re-named *Loemopsylla cheopis* (Rothsch). The others, all of the same genus, are:—

L. pallidus	Taschenb.
L. cleopatræ	Rothsch.
L. nubicus	Rothsch.
L. chersinus	Rothsch.
L. niloticus	Jordan and Rothsch.

I have never seen *P. irritans*, nor have I heard of its being reported from the Sudan, though no doubt it occurs in some districts.

Mention may now be made of Strong's[4] vaccine for plague, which consists of living avirulent cultures of *B. pestis*. He says that a plague culture, which in a dose of two whole agar cultures does not kill a series of guinea pigs, is sufficiently weak to be used for human inoculations, and no plague culture should be so used unless this can be absolutely guaranteed. He tested the action of this vaccine in Manila, commencing with a dose of 1/100 of a loop and going on to the injection of a whole agar culture. The mortality amongst the inoculated was 16·6 per cent. as against 66·6 per cent. among the uninoculated.

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[1] Lamb, G. (May, 1908), "Some Remarks on the Report on Plague, etc." *Indian Medical Gazette*, Vol. XLII.

[2] França, C. Quoted in *Medical Annual*, for 1907, p. 442.

[3] Rothschild, N. C. (1903). *Entomologists' Monthly Magazine*, Second Series., p. 85, Vol. XIV. Quoted in *Medical Annual*, 1907.

[4] Strong, R. P. (June, 1907), "Studies in Plague Immunity." *Philippine Journal of Science*, Vol. II., B. Med. Sciences.

* Article not consulted in the original.

Plague—*continued*

Rogers[1] deals with the blood changes in plague, and quotes Acoyama, who found a very marked leucocytosis present, occasionally exceeding 100,000, most commonly due to excess of polynuclears, but sometimes showing a lymphocytosis instead. There is an occasional absence of leucocytosis, however, in some very severe or very mild cases. The differential leucocyte count was found often to present a peculiarity which may be of diagnostic value. Thus:—

In the leucocytosis of pneumonia and ordinary septic conditions, there is a marked relative increase of the polynuclears, mainly at the expense of the lymphocytes, so that the former number somewhere about 90 per cent., and the lymphocytes only about 10 per cent. In the leucocytosis of plague, on the other hand, the percentage of lymphocytes may not be reduced and may even be actually somewhat high, over 20 per cent. being not infrequently met with, so that the total number of lymphocytes is markedly increased. Unfortunately, this lymphocytosis was only met with in half the cases examined during the first three days of the disease, so that, although its presence is an important aid to the diagnosis, yet its absence in no way excludes the presence of plague. The large mononuclears are not increased, but tend to be low, a point which has served to differentiate plague from malaria.

As regards bacteriological examinations in plague, Rogers says:—

Firstly, a careful search should be made for any primary vesicle on the skin drained by the affected lymphatics in bubonic cases, for these are most frequently met with in the mildest, and therefore the most difficult, cases to distinguish, and they are by no means rare in my experience. If found, the fluid contents should be withdrawn with a sterile syringe after carefully cleansing the skin, and cultures and slides for microscopical examination made.

Secondly, if no primary vesicles are present, but there is enlargement of a group of lymphatic glands, the latter should be punctured in a similar way, in order to ascertain if plague bacilli are present in them, or only staphylococci, as in cases of "climatic bubo." It must, however, be borne in mind that in the suppurative stage of plague, buboes, the specific bacilli, may be absent, and only staphylococci found. Before the suppurative stages, very numerous short bi-polar staining bacilli of plague, will be obtained in this way. These are quite characteristic of the disease, and will enable prompt action to be taken without waiting for the confirmation which will be afforded by the cultivation of the organisms.

Thirdly, in the absence of buboes, either the septicæmic or pneumonic forms of plague may be present. If the former is suspected a small syringe-full of blood should be taken from a vein in the arm and cultures made both on agar and in broth, and films stained and examined microscopically. In the latter stages of the disease, which may be reached as early as the second or third day, the characteristic bi-polar staining bacilli may be present in the circulation in sufficient numbers to allow of their being detected by a microscopical examination and readily isolated by culture. In making cultures from the blood, from 1 c.c. to 2 c.c. should be added to from 50 c.c. to 100 c.c. of broth in a flask to which a few drops of oil may be added in order to obtain the characteristic stalactitic growth of the plague bacillus. E. D. W. Greig, I.M.S., has recently recorded that by this means he cultivated the organism from the blood of 59·8 per cent. of all kinds of plague cases examined soon after admission during the first three days of the disease, the mortality of the positive cases being 97 per cent., while that of the negative was only 43 per cent., so that this method also furnishes evidence of prognostic value.

In pneumonic cases the sputum will show innumerable plague bacilli, often in almost pure culture, and will also allow of the organism being isolated in plates, so a microscopical examination should never be omitted in any case of pneumonia which may possibly be due to plague.

Much work has been done at the agglutination test in the serum diagnosis of plague, but it has not been found to give satisfactory results, and is altogether inferior to the methods above described.

Hossack[2] has a useful paper dealing chiefly with rats and fleas in India. Phenyl is recommended as a pulicide, and a table given comparing it with other disinfectants. References to this subject will be found under "Disinfection" (*page* 44). Ramachandrier,[3] in a very practical kind of paper on prophylaxis, has some quotable remarks on rat destruction. One notes that traps must be of superior quality and must be kept scrupulously clean and free of rat smell. The least smell of the previous day's rats in the cages prevents other fresh rats coming in, hence the traps must be fully immersed in hot water every day and kept in the sun during the whole day. Fresh pieces of mutton or of dried fish constitute the most tempting forms of bait. The virtues of the cat as a rat-killer are extolled in this paper, which concludes with very sound advice to natives on personal precautions, and is altogether well worth careful perusal.

Buchanan[4] was, I think, the first to advocate the keeping of cats as plague preventers, and he concludes a short but interesting paper on the subject as follows:—

The value of a plague prevention measure depends on four things: first, whether it is effective; second, whether it strikes at the root of the disease; third, whether it is available; and fourth, whether it is acceptable to the people.

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[1] Rogers, L. (London, 1908), "Fevers in the Tropics."

[2] Hossack, W. C. (March 15th, 1907), "Rats and Plague." *Journal of Tropical Medicine and Hygiene*, Vol. X.

[3] Ramachandrier, P. S. (August, 1907), "The Prophylaxis of Plague." *Indian Medical Gazette*, Vol. XLII.

[4] Buchanan, A. (October, 1907), "Cats as Plague Preventers." *Indian Medical Gazette*, Vol. XLII.

Plague— *continued*

There is now not a shadow of a doubt that the rat is the cause of plague epidemics, and no evidence is required to convince most people, and especially the natives of this country, that the best way to get rid of rats is to keep cats. Even if isolation, disinfection and inoculation were of much value, they do not strike at the root of the disease. Rat destruction by traps and poison is troublesome and costly, though effective if the staff employed on this work is continuously energetic, but the cat requires no stimulus.

That the cat is available, or can soon be made available in sufficient numbers in this district, is clear from the census that has recently been taken, and from the fact that there is the greatest willingness on the part of the people to obtain young cats and keep them.

The strongest point in favour of the cat is—a fact which very few Europeans in this country know—that it is almost a religious duty with a Mohammedan to keep a cat, and that Hindus consider it a great sin to injure even a single hair of a cat. It is now recognised that the success of measures for plague prevention depends very greatly on the attitude of the people towards it. The strongest point in favour of recommending the keeping of cats is that any prejudice that is likely to be met with is in favour of, and not against, the keeping of cats, excepting in the case of the Bhowan Dhers and a few Jains (and their numbers are not large). It is clear then that the cat is effective; that it strikes at the root of the disease; and in this district it is available, and that it is acceptable to the people. During the past ten years I have seen many measures introduced for plague prevention. To all there has been more or less opposition. To the keeping of cats there is practically no opposition, and it is clear that the keeping of cats is *par excellence* the people's remedy for plague prevention.

An interesting suggestion is that made by Saigol[1] to the effect that rats and guinea pigs should be used as "plague barometers." His argument is that if these rodents are kept in the house, fleas leaving an infected dead rat will attack them in preference to human beings. He suggests several cages to each household, each containing at least three rats. Guinea pigs also attract *P. cheopis*. These animals will then act: (1) As "plague barometers"; (2) as flea-catchers and retainers—flea traps, in short; (3) as "locality indicators." Saigol notes that the method cannot be said to be unnecessary, because it is well known that on the appearance of an epizootic amongst rats all the healthy ones quit the locality leaving their sick and dying behind. These latter are the source of infection. "Barometer rats," being unable to escape, would indicate the presence of the disease.

The same author[2] has recently recorded his experiments with the Danysz virus, "ratin," "azoa," "common sense exterminator," and sulphur fumigation. In no instance have the results been entirely satisfactory, and, as regards rat destruction on a large scale, the outlook is at present none too hopeful.

Colvin[3] has a paper on the latency of plague and its spread by means of fomites, citing cases in connection with the Glasgow epidemic. There seems to be no doubt that in these instances the infected clothing conveyed the contagion. He concludes with the following quotation from Simpson's well-known treatise on plague:—

There is one feature of the present pandemic of plague that presages danger in the future. It is that notwithstanding its apparent inability to cause in one place a great epidemic, it exhibits in some places marvellous powers of recrudescence and resistance to all known measures of prevention, and this even when the cases are few. This tenacious capacity, combined with its transportability, makes it formidable because its slow progress, few cases, and possibly slight mortality accustom the people to its presence and lull the authorities into a frame of mind of looking upon it as a disease that can be easily controlled. In the meantime it gradually dots itself over different parts of the country, securing a firm hold in some localities, which again form fresh centres for its activity, until in the course of a few years it is firmly established in the country at many centres and only awaits the conditions necessary for its development into an alarming epidemic.

Thompson[4] deals with experiences in Sydney, and is of opinion that the exclusion of rats from occupied buildings in cities is the only measure which can permanently diminish the susceptibility of India to plague. This, as he points out, is the true preventive method, the use of the prophylactic, evacuation of infected places, and rat destruction being remedial.

Something has already been said regarding the disinfection of ships infected with plague and infested with rats (*see* "Disinfection," *page* 44), but a note on a paper by Bonjean[5] may be quoted:—

In 1903 the International Conference, meeting at Paris, indicated three processes: (1) an admixture of sulphurous acid with a small quantity of sulphuric acid; (2) a mixture of the monoxide and dioxide of carbon; and (3) carbon dioxide. Of these methods the first is destructive to insects and bacteria as well as to the rodents, At Hamburg the toxic gas consists of carbon monoxide, 5 volumes; carbon dioxide, 28 volumes; and nitrogen,

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[1] Saigol, R. O. (December, 1907), "Rats and Guinea Pigs as 'Plague Barometers' *v.* Rat Destruction." *Indian Medical Gazette*, Vol. XLII.

[2] Saigol, R. O. (July, 1908), "Rat Extermination." *Indian Medical Gazette.*

[3] Colvin, T. (November 30th, 1907), "Is Bubonic Plague still lurking in the City of Glasgow?" *Lancet*, Vol. II.

[4] Thompson, J. A. (December 21st, 1907), "Protection of India from Invasion by Plague." *British Medical Journal*, Vol. II.

[5] Bonjean, E. (March 21st, 1908), "Means Employed for the Destruction of Rats on Ships." *Lancet*, Vol. I.

77 volumes. This process effectually kills the rodents without injuring the merchandise carried on vessels, but it has disadvantages. It requires costly apparatus and a prolonged exposure to the gases—not less than 24 hours. Further, it is dangerous to man, while it does not destroy insects. The use of carbon dioxide alone has been abandoned owing to its high price. In France preference is given to a mixture of sulphurous and sulphuric acid gases, obtained by the free combustion of sulphur in air, but M. Bonjean's experiments have shown that sulphurous acid alone, in the proportion of 60 to 80 grammes of the gas per cubic metre, destroys rats almost as quickly without exciting so destructive an action upon merchandise. It is important to avoid humidity in the air as much as possible and to drive out the residual gas after the rats have been killed. In actual practice the gas may be produced by the Marot apparatus, using 70 grammes of liquefied sulphurous acid per cubic metre, or by the Clayton or Gauthier-Deglos apparatus, using 35 grammes of sulphur per cubic metre. After two hours the remaining gas is removed by ventilators and the dead rats are burned or thrown overboard.

Plague— *continued*

Leishman[1] records the views recently advanced at Berlin. Giemsa held that it was absolutely necessary that all ships which had anchored at plague-infected ports, or had taken cargo from them, should have all the rats destroyed by some sure means, so that none should be free to get ashore at a new port and infect the rats of that port. The ideal gas for this purpose has not yet been discovered, but, after mentioning the disadvantages of liquid carbonic acid gas and sulphurous acid as used in the Clayton process, he recommended that already mentioned under "Disinfection" (*page* 44), and which consists of carbonic oxide 5 per cent., carbonic acid 18 per cent., and nitrogen 77 per cent. Its advantages are—(1) it is devoid of smell and of chemical activity, and is thus little likely to damage cargo; (2) it is very deadly to rats and certain to kill all of them on board; (3) it is relatively cheap and can easily be produced in very large quantities, so that even large ships can be filled with the gas in a few hours.

Tjaden pointed out that its failure to kill fleas constituted a grave defect, and thought that in many cases a sulphur process might be preferable.

There is little to be said about the treatment of plague, which still remains more or less symptomatic, but Elliot[2] speaks well of collargol, an allotropic form of silver. It is soluble in water about 1 in 25. He used it in intravenous injections, 1 c.c. of a 1 per cent. solution, and he thinks the drug merits further trial.

Choksy[3] writes on recent progress in the serum-therapy of plague and concludes that in Yersin-Roux anti-plague serum we possess a useful and efficacious remedy against plague. He points out that *the absence of all antitoxic action* is responsible for its limited utility. The whole secret of the treatment lies in applying the serum very early. Indeed, after the lapse of 48 hours it does not appear to influence the course of the disease perceptibly. It would seem also that the action of the serum is more beneficial after protective inoculation.

Relapsing Fever. *See* "Spirochætosis" (*page* 185).

Scorpion Sting. Scorpions are common in the Sudan, and cases of scorpion sting by no means rare. Professor Werner, of Vienna, has kindly furnished me with the following list of the species of Sudanese scorpions identified up-to-date by Dr. A. Birula of St. Petersburg.

1. *Prionurus (Buthus) amoureuxi*, Savigny; the big, yellow, thick-tailed scorpion of the Northern Sudan deserts. The crests of the upper side of the last segments of the tail are toothed and strongly raised. In the next species they are smooth and feebly raised.

2. *Buthus quinquestriatus*, Hemps and Ehrenbg.; the smaller, yellow species with the tail more or less blackish at the end. It is the most common species, and is found in gardens in Khartoum.

3. *Hottentotta (Buthus) minax*, C. L. Koch; a brownish species found in the Southern Sudan on the White Nile.

Some statistics regarding scorpion sting in the Sudan are available. Bray in 1902 reported twenty-one deaths from this cause: the age distribution being, under 1 year, five; from 1 to 5 years, nine; from 5 to 15 years, seven.

In 1900, forty cases of deaths were known, one being that of a young adult aged 18 years. In the 1898 Sudan campaign, a British soldier was killed by a scorpion which stung him several times in the back.

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[1] Leishman, W. B. (April, 1908), "The International Congress of Hygiene, Berlin, Sept., 1907." *Journal of the Royal Army Medical Corps*, Vol. X.

[2] Elliot, A. M. (August 17th, 1907), "Some Remarks on Plague." *Lancet*, Vol. II.

[3] Choksy, N. H. (May 30th, 1908), "On Recent Progress in the Serum-Therapy of Plague." *British Medical Journal*, Vol. I.

Scorpion Sting— *continued*

Death above 15 years of age is said to be rare, but in 1907, at Omdurman, there were two fatal cases out of a total of eighty-eight occurring in persons above this age.

Altogether 167 cases were treated in the Civil Hospital, of which eleven terminated fatally.

Wilson[1] is the author of a paper on the venom of Egyptian scorpions. The three common species in Egypt are *Buthus quinquestriatus*, *Prionurus citrinus* and *Buthus maurus*, a black scorpion. The paper is chiefly of a physiological nature and evidences much careful work. The chief symptoms in man are given (*vide infra*), and others more rarely seen—such as hallucinations and mental disturbance, sometimes convulsions, weak pulse, rapid respiration, etc. Local redness and swelling may be present, but often the local reaction amounts to no more than would be caused by a mosquito bite.

A list of animals immune to the venom are given, amongst which one notes a species of gerbil and of jerboa. One tabulates here such of the general conclusions as are most likely to be of value :—

The venom is a clear, slightly viscid fluid of faintly acid reaction and high specific gravity. It contains from 20 to 28 per cent. of solids of which inorganic salts form a considerable part. Proteids in part coagulable by heat, are present and probably form the most important organic constituent. The characters of the venom appear to differ in different species.

The active principle of the venom is apparently of a proteid nature. So far my investigations tend to show that it is either a nucleo-albumin, acid-albumin, or a primary albumose.

The active principle is soluble in glycerin and in saline solution; it is probably insoluble in pure water and is insoluble in 85 per cent. alcohol.

The active principle is unaffected by drying. When in solution it is very resistant to putrefaction, and is not affected by heating to 100° C. for short periods, but is destroyed if the heating is continued for from 12 to 15 minutes.

The minimum fatal dose of the toxin for the guinea pig is approximately 1 mg. per kilogramme. The toxic value is, therefore, 10,000,000.

The amount of toxin contained in the glands of different species of scorpion appears to differ. Of the three species examined, most could be obtained from *Buthus quinquestriatus*, least from *Prionurus citrinus;* the former is therefore to be regarded as the most venomous.

The symptoms in animals are referable to the muscular and glandular tissues, consisting of muscular spasms, copious secretions and apparent muscular paralysis.

The muscular spasms are of peripheral origin and are due to the direct exciting action of the poison and to the increased effect of stimuli upon the contractile tissue. The paralysis is due to the great depression in the excitability of the muscles to direct and especially indirect excitation resulting from the fatigue brought on by the action of the poison.

Death is mainly due to the inability of the respiratory muscles to respond to stimuli reaching them from the central nervous system. There also appears to be a direct action on the pulmonary tissues, the effect of the poison being possibly to produce spasm of the smooth muscle of the bronchi and pulmonary blood-vessels.

The effects of scorpion venom in man are comparable to those seen in the guinea pig, the convulsive symptoms being, however, much less prominent. Local pain, vomiting, sweating and salivation are the most marked symptoms.

Death from scorpion sting is very rare in persons above the age of fifteen years, but occasionally occurs in adults.

Certain animals living under conditions which render them liable to the attack of scorpions are immune to the venom.

Nicolle and Catouillard[2]* experimented with the venom of *Heterometrus maurus*, a common North African species, with a view to finding an antidote or a protective serum, but their results were disappointing. I understand a more promising research is now proceeding in Cairo. Antivenin, as is to be expected, has no protective action, as scorpion poison does not seem to resemble any form of snake poison. Some persons, fakirs and others, have a curious power of handling scorpions with impunity, although stung by them, so that there must be some way of producing immunity.

Simpson[3] recommends a paste of ipecacuanha as a local application in cases of scorpion sting.

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[1] Wilson, W. H. (1904), "On the Venom of Scorpions." *Records of Egyptian Government School of Medicine*, Vol. II.

[2] Nicolle, C., and Catouillard, G. (1905). *C. R. Soc. Biol.*, t. LVIII, 231–233.

[3] Simpson, W. J. (1905), "Maintenance of Health in the Tropics." London.

* Article not consulted in the original.

Scurvy. Coplans,[1] in a paper on Scurvy in South Africa, endeavoured to show that the disease was due to an infection for which food may act as a vehicle under conditions of dirty storage or dirty preparation, and that its infectivity depended on the insanitary habits, the degree of intimacy, the overcrowding and, perhaps, the unwholesome occupation of those who were its victims. In the discussion on this paper, Wright upheld the view that scurvy is neither a bacterial disease nor that it is associated with dirt, but that it depends upon a diminution in the alkalinity of the blood. Hence the administration of potatoes, fruits, raw meat juice, etc., is scientifically justified as supplying all the elements which are turned into alkalies in the body. A much more rapid result can, however, be obtained by the exhibition of lactate of soda, lactic acid being the acid most easily converted into carbonic acid in the body. Lime juice ought to be eschewed as containing citric acid, which can only decalcify the blood, and is useless as an anti-scorbutic, because it breaks up into CO_2 and H_2O without leaving any residue. The quantity of citrates in lime juice is too small to be of any value. The rest of the discussion, though interesting, yielded little of definite value. Kenwood thought that infantile scurvy was probably a different disease from that described, and Willoughby cited the theory of Tonk ! *(sic)* of Christiania, who believes scurvy to be simply a chronic ptomaine poisoning. This, it is said, is the only theory which adequately explains all cases of the disease. (*Vide infra*)

A different view is that taken by Barnardo[2] as the result of experience in Somaliland. He is inclined to think that scurvy is a toxæmia, due not to any new specific organism but to the bacteria normally present in the alimentary canal which have taken on an abnormally active development and toxin formation consequent on the diminished vitality of the mucous membrane there, and a diminished bactericidal power of the tissues generally. This, if true, indicates the exhibition of intestinal antiseptics. Barnardo agrees with Wright as regards the blood change and the uselessness of lime juice. He regards pickles as a useful addition to the dietary, and recommends fresh meat juice combined with potassium citrate and calcium chloride.

An interesting paper is that by Ekelöf,[3] on the Swedish Antarctic Expedition, wherein he, like Willoughby, alludes to the theory of Torup (not Tonk) of Christiania, as to scurvy being a chronic ptomaine poisoning due to the introduction into the body of some non-organised matter, foreign to, and dangerous for, the human organism. This must be of the nature of a poison due to a peculiar and slow decomposition taking place in badly preserved (*e.g.* badly salted) meat and fish. While Ekelöf thinks this is the only theory giving an intelligible explanation which, in its leading features at least, is compatible with the actual circumstances, he is careful to point out that it cannot be true in its entirety, for scurvy has attacked expeditions furnished with provisions of first-rate quality.

Holst and Frölich,[4] already quoted in connection with beri-beri, conducted feeding experiments on guinea pigs, and their results may be tabulated :—

A one-sided diet consisting of various sorts of grain, groats and bread, produces in guinea pigs a disease that corresponds, macroscopically as well as microscopically, to human scurvy.

On the other hand, they found that this disease does not occur after a one-sided diet consisting of fresh cabbage or fresh potatoes, whereas it again is produced by dried potatoes. That is, the disease originates in guinea pigs as well as in man as a result of a diet confined to some special nutriments.

They have further observed that the disease is favourably influenced by different sorts of nutriments known, from human experience, as "antiscorbutics." They have, however, also found that at least one of these nutriments, that is cabbage, loses a deal but not all of its preventive power when boiled for half-an-hour at 110° C.

Finally, they quoted several examples showing that the same or similar one-sided diets that produce the disease in guinea pigs have repeatedly produced scurvy in man.

However, they have not, hitherto, been able to produce the disease that has been the proper aim of their experiments—that is, the younger brother of scurvy or ship beri-beri—

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[1] Coplans, M. (1903-4), "On the Etiology of Scurvy." *Trans. Epid. Soc.* London, pp. 79–94.

[2] Barnardo, J. F. (July, 1904), "Scurvy in Somaliland." Notes on the Condition of the Blood Serum. *Indian Medical Gazette*, Vol. XXXIX.

[3] Ekelöf, E. (October, 1904), "Medical Aspects of the Swedish Antarctic Expedition, Oct., 1901–Jan., 1904." *Journal of Hygiene*, Vol. IV.

[4] Holst, A., and Frölich, T. (October, 1907), "On the Etiology of Scurvy." *Journal of Hygiene*, Vol. VII.

Scurvy— *continued*

though they have repeatedly, in guinea pigs, seen abortive cases of scurvy recalling the latter disease. This problem is, however, by no means clear, nor have they hitherto been able to make experiments explaining, in an unmistakeable way, why the one-sided diets mentioned above produce scurvy.

Okada and Saito[1]* found a micrococcus in the blood of scurvy patients, and regard this as the actual cause of the disease.

MacVicar[2]* came to the following conclusions from observations made on natives in Cape Colony :—

1. The disease common amongst the natives of his district, known to them as "umtshetsha," is true scurvy.

2. The main cause of the disease is the absence from the diet of fresh food, animal or vegetable. This in some cases was not entirely absent, but present in the diet in insufficient quantity.

3. Muscular exertion precipitates scurvy. Thus, in several cases, though the patients had been living at their homes on a diet lacking in the fresh element, they showed no marked symptoms of scurvy till they commenced active work.

4. Food may be in perfectly good preservation and yet lack the anti-scorbutic power of "fresh" food. Thus, men on polar expeditions using tinned vegetables and even lime-juice, became scorbutic, but lost their symptoms of scurvy as soon as they had access to "fresh" seal-meat.

Scurvy visited the old civil prison, Khartoum, and was present in a severe form, simulating beri-beri and causing one death. As to the cause, it is very difficult to come to any conclusion, though one is inclined to favour Barnardo's view. The diet was certainly of a monotonous nature, and rapid disappearance of the symptoms occurred when fresh milk and beans were added to the dietary together with melons, and when oral hygiene received proper attention.

Sewage. It is, of course, impossible to enter at all fully into so huge a subject, and in any case certain aspects of it fall to be discussed under "Sanitary Notes" (Third Report). Here we propose merely to indicate some general papers of special interest to those working in the Tropics, and to consider others containing points not commonly found in text-books. It may be said at once that one of the most useful little works on sewage disposal in the Tropics is the essay by Major Smith.[3] It does not lend itself to quotation nor are the conditions mentioned altogether those prevailing in the Northern Sudan. The work deals with true, humid, tropical regions where the rainfall is heavy and problems are more difficult than in dry and desert districts. Much, however, applies forcibly to the posts in the Southern Sudan and will repay careful study.

An excellent paper is that by Cornwall[4] on the system of drainage and sewage (domestic and municipal) best suited for tropical climates. The various systems are briefly but adequately considered, and their advantages and disadvantages discussed. One has found this article of great service on several occasions.

Of the same class, but more extended, are the papers by Simpson[5] on tropical hygiene, who notes that the secret of success in any conservancy system in the Tropics is the protection from rain and floodings.

Of books, "Drainage Problems of the East," by James; "The Purification of Sewage," by Barwise; and the latest edition of a work on the same subject, by Dibdin, will be found of value.

A useful, illustrated description of the Liernur system will be found in the *Lancet* of July 11th, 1908.

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[1] Okada, K., and Saito, Y. (1907). Quoted in *Medical Annual*, 1907, p. 493.

[2] MacVicar, N. (1907). Quoted in *Medical Annual*, 1907, p. 494.

[3] Smith, F. (London, 1904), "Drainage, Sewerage and Conservancy in Tropical Countries and Elsewhere."

[4] Cornwall, J. W. (August 1st, 1903), "The System of Drainage and Sewerage (Domestic and Municipal) best suited for Tropical Climates." *Journal of Tropical Medicine*, Vol. VI.

[5] Simpson, W. J. (December 15th, 1903, and January 1st, 1904), "Collection, Removal and Disposal of Waste Waters." *Journal of Tropical Medicine*, Vols. VI. and VII.

* Article not consulted in the original.

Sewage—*continued*

An interesting account[1] is given of the successful working of a sewage farm (septic tank system) in India. It served a leper asylum which contained fifty lepers; the whole of the effluent was used on a farm of about five-and-a-half acres, and the produce from the farm amounted to thirty-six tons per acre. Plantains, guinea grass, lucerne grass and vegetables were grown, but karbi and jowar (*dura*, millet) constituted the chief products. (In this connection one may note that there is no evidence to show that the growing of fruit on sewage farms is injurious to health, *vide Public Health*, December, 1906.)

The gas given off from the septic tanks worked an engine, which actuated a small centrifugal pump and daily lifted as much of the fluid to the filters as was required. The fodder crops were used for the feeding of bullocks, and the profits wholly maintained the fifty lepers.

When the effluent, as in this case, is chiefly applied to the land, one purification only of the sewage appears necessary, and nearly the whole of the effluent used on the farm came directly from the septic tank. Unfortunately, the cost of establishment and of maintenance is not mentioned.

Caldwell[2] contrasts artificial and natural disposal of sewage. He points out that the three principal methods now employed are the chemical, the biological and the natural—the first two being classed under the term "artificial." He speaks strongly in favour of the natural methods, especially when shallow trenches are used as in some parts of India, saying:—

"The contents of latrine buckets and night-stools are collected by the sweeper, dumped into a Crowley's filth cart, and carried to the grass farm, where the contents of each cart, averaging sixty gallons, are emptied into a trench 9 inches deep 5 feet wide and 16 feet long. The trench is then filled in, and in the course of a few days all trace of offensive matter has, under the influence of the soil organisms, absolutely disappeared. It is an interesting fact, as bearing on the presence of the necessary organisms in the superficial layers of the soil only, that a horse, galloped over ground newly operated on, could not make an impression in the soil deeper than two inches, whereas if refuse was buried at a greater depth horses sank almost knee-deep in the soil when taken over it. I have myself examined samples of soil carefully by ordinary bacteoriological methods, and I have never succeeded in detecting the presence of coliform bodies later than the fifth day after burial, but I must at the same time make it absolutely clear that I have no intention to dogmatise concerning a matter which certainly needs further investigation."

This question will be discussed more fully under "Sanitary Notes" (Third Report).

Kenwood,[3] in criticising a paper by Starkey on the economical disposal of sewage in small rural communities, draws attention to the danger of sewage becoming over-septicised if left too long in a septic tank. The result is a deposition of colloid matter which renders the working of the tank very ineffective.

Phelps and Winslow[4] advocate the use of methylene blue in testing sewage effluents, *i.e.* as regards their stability with reference to putrefaction. They state that it directly measures the quality of most importance in a sewage effluent, *i.e.* its freedom from the tendency to putrefactive change, and that it registers this with a greater delicacy than any method with which they are acquainted. Moreover, it is so simple in technique as to be peculiarly adapted to sewage works where neither a laboratory nor an expert chemist are available. Their description of it is as follows:—

A small portion of an aqueous solution of the dye (in our experiments 1 c.c. of a 0·1 per cent. solution) is added to the effluent in a glass stoppered bottle (250 c.c. capacity in our work), and the sample is then incubated either at 20° C. or at 37° C. The blue colour of the solution remains practically unchanged during the period of observation until the available oxygen contained in it is used up and putrefactive conditions arise. At this point the dye is reduced and decolorised. The time required for such decolorisation is a quantitative measure of the degree of putrescibility of the sample and the retention of the colour for a period of one week or more, at 20° C., or of four days at 37° C., may be taken as an indicator of its stability.

Clemesha,[5 6] in two papers considers the value of chlorinated lime for sterilising septic tank effluents in India, in order to prevent the pollution of rivers, and concludes:—

(i.) That 5 grains per gallon of chloride of lime is ample to sterilise a bad effluent, and that this amount leaves a good margin for erratic working.

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[1] "A Successful Septic Tank and Sewage Farm: Notes from India." *Lancet*, June 4th, 1904, Vol. I.

[2] Caldwell, R. (March, 1908), "Artificial and Natural Sewage Disposal Contrasted." *Journal of the Royal Institute of Public Health*, Vol. XVI.

[3] Kenwood, H. R. (August 17th, 1907), Meeting of Brit. Med. Assoc. Sec. of State Medicine. *Lancet*, Vol. II.

[4] Phelps, E. B., and Winslow, C. E. A. (May, 1907), "Use of Methylene Blue in Testing Sewage Effluents." *Journal of Infectious Diseases*, Suppl. 3.

[5] Clemesha, W. W. (March, 1906), "Experiments for Sterilising Septic Tank Effluent." *Indian Medical Gazette*, Vol. XLI.

[6] Clemesha, W. W. (October, 1906), "Further Note on the Use of Chloride of Lime to Sterilise Septic Tank Effluents." *Ibid.*

Sewage— *continued*

(ii.) That 1 grain per gallon of chloride of lime is sufficient to sterilise an effluent, provided the lime contains over 30 per cent. available chlorine, that it is added in the best method, and that the effluent is a good one.

(iii.) That sunlight is the powerful factor in splitting up the unstable compounds of oxygen, hydrogen and chlorine.

(iv.) That a weak solution of chloride of lime is altered with extraordinary rapidity in the sunlight; the stronger the solution the less it is altered.

(v.) That even weak solutions lose very little of their available chlorine if kept out of the sun.

(vi.) That the best method of adding chloride of lime to an effluent is to make a mixture of the powder in water and run in the liquor. For this process it is necessary to make up a strong solution, six or twelve ounces per gallon, and keep the lid on the receptacle.

(vii.) That a weak solution of chloride of lime so rapidly oxidises in the sun that free chlorine cannot be found in the river a short distance from the drainage outfall.

(viii.) That the chloride of lime kept under suitable conditions does not deteriorate so rapidly as to render the process liable to failure.

Discussing possible objections to this method, he says that even the best effluent contains millions of organisms per c.c., and that in India, where there are small installations and a very concentrated sewage, which is practically wholly human excrement, it is advisable, when it can be done cheaply, to sterilise the effluent. The objection that chlorinated lime is very unsuitable, and rapidly deteriorates in a hot country, can (he says) be met with a little arrangement. The papers are interesting, but one is inclined to think that in most districts proper application to land would be preferable. Further information on this subject will be found in a review of a paper by Korpjuiweit.[1]*

O'Meara[2] furnishes a useful paper on sewage purification in India generally and Mirzepur in particular, discussing septic tanks, continuous filters *versus* contact beds, and several other subjects. As he tabulates his conclusions in each section his results are easily followed.

Skin Diseases. Under this heading we deal with recent work on these complaints as they present themselves in the Tropics or in sub-tropical countries.

Madden[3] has a paper on "Nile Boils in Egypt." These appear during the hot months and especially in the damp weather towards the end of the summer, and chiefly attack European residents, though the native Egyptian is by no means immune. The Nile boil is an inflammatory and exceedingly painful condition, which does not last more than fourteen days from beginning to end. The favourite site is the perinæum, especially all round the anus, the buttocks, the arms, the hands, the fingers and the face. The boils are more common in men than in women, but are no respecters of age or sex, though happily rare in young children.

Madden describes the boil originating in a hair follicle causing swellings, severe throbbing and pain and inflammatory implication of the neighbouring lymphatics. The surrounding zone of redness, the tiny central blister and the pea-green sloughs are very typical. The swollen area may be very large. Once the "core" is evacuated the cavity rapidly heals. A culture from the slough yielded a pure growth of *Staphylococcus pyogenes aureus*. Treatment, he notes, is very unsatisfactory, and consists chiefly in fomentations and expectancy. Madden thinks the old "Bouton-de-Nil" does not now exist in Egypt, and that its place has been taken by this acute inflammatory condition, which he regards as a distinct entity.

Elliott[4] follows with a paper on "Natal Boils," but he seems to mix up true Oriental sore with the simpler inflammatory conditions. He thinks conditions of tropical life combine to lower vitality and render residents liable to such septic outbreaks. In Natal sores, if the slough does not come away or is not removed, keloid results. He speaks very highly of the value of quinine, which in his own case, although chronic, acted as a specific. Unfortunately, he does not give the dosage. In Khartoum I have seen a case in which small boils occurred in crops, chiefly behind the ears, recurring nearly every year and in many ways resembling the Nile boil in miniature. They were not benefited by quinine.

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[1] Korpjuiweit, O. (January, 1908), "The Treatment of Sewage with Chloride of Lime." Quoted in *Journal of the Royal Institute of Public Health*, Vol. XVI.

[2] O'Meara, E. J. (July, 1908), "Notes on Sewage Purification." *Journal of the Royal Institute of Public Health*, Vol. XVI., No. 7.

[3] Madden, F. C. (October 1st, 1906), "Nile Boils." *Journal of Tropical Medicine and Hygiene*, Vol. IX.

[4] Elliott, J. F. (January 1st, 1907), "Natal Boils." *Journal of Tropical Medicine and Hygiene*, Vol. X.

* Article not consulted in the original.

Skin Diseases—*continued*

Ashley-Emile[1] describes Zambesi ulcer, which is of a phagedænic nature, and which has been supposed to be many things. He states that it is due to myiasis, being produced by the larva of a fly, which is certainly not that of *Bengalia depressa*. After reading the paper, one comes to the conclusion that *Cordylobia anthropophaga* is to blame. (*See* "Myiasis," *page* 137.)

Shattuck[2] has notes on chronic ulcers occurring in the Philippines. He describes four types and discusses their etiology, but without coming to any definite diagnosis.

Of greater interest is a peculiar skin disease described by Ziemann[3]* as affecting the negroes on the West Coast of Africa. It is a leucoderma, and apparently something like it exists in British Central Africa. Ziemann says:—

> The disease is characterised by the appearance of round, oval, or irregularly-shaped patches of a bright, pale, yellowish-red colour on the skin of the extensor and flexor surfaces of the hands and feet. The predominant colour of the affected areas is of a whitish tinge, and the general aspect may be compared to that of extensive superficial burns which have healed without the formation of granulation tissue. The disease begins at the age of from 10 to 15 years; its course is symmetrical, either attacking the hands first and then the feet, or the feet first and the hands afterwards, or both at the same time. The progress of the disease is extremely chronic.

Dr. Ziemann has never seen more than four-fifths of the surface of the hands or feet affected with pigment atrophy, and at this stage the process appears to have terminated. The flexor surfaces appear to be more extensively affected than the extensor. Another characteristic of the disease is that the pigment atrophy is practically confined to the hands and feet, only a slight further extension being noticeable after the lapse of ten, fifteen or twenty years. There is a gradual and more or less complete loss of pigment in the hairs situated in the atrophied regions. Sensibility appears to be normal; at least, Dr. Ziemann has not been able to ascertain any impairment in the sensations of either touch, temperature, or pain; nor was there any alteration in the muscle sense. The sweat glands in the affected areas were normal. The electrical reactions of the affected limbs were normal, and there was no evidence in any part of the body of any lesions affecting either the nervous system, the blood, or the excretory functions.

Leucoderma is not uncommon in the Sudan, but seems to be generally regarded as most frequently the result of syphilis.

Cantlie,[4] in a general and suggestive paper to be considered later, remarks:—

> The condition I have termed "foot tetter," for want of a better name, as the exact pathology of this disease is not yet made out, is a troublesome affection of the soles of the feet, characterised by areas of raised epidermis, which become scaly and hard; cracks and fissures appear about the toes, causing at times lameness, and attended by intense itching. The disease is very persistent, and may last for years—ten or even more after the patient has left the Tropics, recurring during hot summer weather in this country, although never wholly dying out during the winter.
>
> The treatment is to completely cover the affected parts with some paste or plaster which will wholly exclude the air for several weeks; as this treatment is troublesome and inconvenient, few will be found to carry it out.
>
> Of the many skin affections, most are common to both temperate and tropical countries, although a few are part and parcel of some general condition of tropical origin. Perhaps the most interesting of these, and the most difficult to rightly interpret, are the skin lesions found in sleeping sickness. Strange as it may seem, cases of this disease do occur in white men coming to Britain, and the peculiar wandering, irregular, eczematous-looking patches presented by the skin would be impossible, in the present state of our knowledge, to explain aright, had we not other symptoms and signs to guide us.

Molluscum contagiosum occurs in the Sudan. I recall a case in an Egyptian soldier, and examining the growth histologically. The etiology of the condition still seems obscure.

Casagrandi,[5]* after filtering the ground mass of human molluscum through a Berkefeld bougie, on examining the fresh filtrate found small motile corpuscles elongated or pear-shaped, while on staining by Giemsa he found two different elements, the one taking on a carmine-red colour, the other a blue tint. The latter are the least interesting. The red elements, which seem to possess a granular structure, are possibly protozoa.

Lipschütz[6]* found different elements which appear to be identical with those described by Borrel. They are immobile in the fresh state and do not possess cilia. Stained by Giemsa for two hours these elements appear as masses of small, homogeneous granules, which may occur singly, in the form of diplococci, or as short chains like streptoccoci.

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[1] Ashley-Emile, L. E. (September 15th, 1905), "Zambesi Ulcer." *Journal of Tropical Medicine and Hygiene*, Vol. VIII.

[2] Shattuck, G. C. (December, 1907), "Notes on Chronic Ulcers in the Philippines." *Philippine Journal of Science*, Vol. II., B. Med. Science.

[3] Ziemann, H., *Arch. für Derm. und Syph.*, Bd. 74, Heft. 2, No. 3. Quoted in *British Medical Journal*, May 13th, 1905, Vol. I.

[4] Cantlie, J. (June 22nd, 1907), "Clinical Observations on Tropical Diseases as they are met with in Britain." *British Medical Journal*, p. 1468, Vol. I.

[5] Casagrandi, O. Quoted in *Bull. de l'Institut Pasteur*, January 15th, 1907, Vol. V.

[6] Lipschütz, B. (1907). *Wien. Klin. Woch.*, t. XX., No. 9.

* Article not consulted in the original.

Skin Diseases— *continued*

Prowazek[1] has suggested the title Chlamydozoa to include the pathogenic micro-organisms said to be the cause of a group of maladies, *i.e.* variola, vaccinia, scarlatina, hydrophobia, avian plague, trachoma, molluscum contagiosum, contagious epitheliomas of birds, and some others. They are intracellular microbes and approach the protozoa in certain particulars.

Castellani[2] discusses the tropical forms of pityriasis versicolor. Several forms exist, the two chief being the yellow and the black. The yellow, *Pityriasis versicolor flava* is the commonest. A *Pityriasis alba* also occurs, in which the fungus is very abundant. It is easily cured, unlike flava. *Pityriasis nigra* usually attacks natives alone. Castellani terms the fungus of *P. flava*, *Microsporon tropicum*, and gives a drawing of it as of *M. mansoni*, the fungus found in *P. nigra*. He called the fungus of *P. alba*, *M. macfadyeni*. Its mycelium and spores are of very small dimensions. He succeeded in growing *M. mansoni*, and gives its cultural characteristics on various media. What is said regarding prognosis and treatment may be quoted :—

None of the tropical forms of *Pityriasis versicolor* show any tendency to spontaneous cure. All the forms are very chronic and may last for life. The forms yielding most readily to treatment are *Pityriasis nigra* and *Pityriasis alba ;* the most obstinate is *Pityriasis flava.* For *Pityriasis nigra* and *alba*, the usual antiparasitic lotions and ointments answer well ; a salicylic spirit lotion (4 per cent.) followed by a mild mercurial ointment as, for example, white precipitate, gr. 10 to gr. 15 to the ounce of vaseline, gives good results. *Pityriasis flava* is much more difficult to deal with ; turpentine applied every day and followed by a beta-naphthol or epicarin ointment is often successful, but the treatment must be continued for months. It is to be noted that in several cases of *Pityriasis flava* the fungus has apparently a deep, permanent disturbing action on the pigmentation processes of the skin, because even where the fungus has been destroyed the patches remain of lighter colour than the surrounding skin for a long time, though ultimately they become normally pigmented.

One knows next to nothing regarding these infective skin conditions in the Sudan. Probably the Northern Sudan has so dry a climate that their development and growth are not favoured. This is one of the many subjects on which information is desired.

While prickly heat (miliaria) does not frequently afflict residents in Khartoum, I understand that sometimes it causes much suffering amongst those stationed in the more humid southern regions of the Sudan.

The following prophylactic treatment[3] may, therefore, be noted :—

Wear thin, light woollen garments next the skin, expose the body to heat as little as possible, avoid constipation, and apply the following lotion locally :—

Acidi carbolici,		℥ ss
Acidi boracis,		ʒ i
Zinci oxidi,		ʒ iss
Glycerini,		ʒ ii
Alcoholis,		ʒ ii
Aquæ *q.s.*	ad.	℥ vi

and a dusting powder consisting of—

Magnesii carb., acidi borici, pulv. amyli, etc. āā ʒ ii.

When the entire body is involved, bran, starch or alkaline baths are indicated. Hyde recommends :—

Acidi carbolici,		ʒ i
Glycerini,		ʒ ii
Mentholis,		ʒ i
Sp. vin. rectif.,		℥ i
Aquæ *q.s.*	ad.	℥ viii

as a local application.

Pearse[4] believes that in prickly heat the sebaceous glands are primarily at fault, and states that their own secretion causes an acute obstruction which then is secondarily

[1] Prowazek, S. (April 30th, 1908), "Chlamydozoa." *Bull. de l'Institut Pasteur*, Vol. VI.

[2] Castellani, A. (February 15th, 1907), "Observations on Tropical Forms of Pityriasis Versicolor." *Journal of Tropical Medicine and Hygiene.*

[3] Quoted in *Journal of Tropical Medicine and Hygiene*, August 1st, 1906, p. 241.

[4] Pearse, T. F. (May 23rd, 1908). *Lancet*, p. 1489.

maintained by the continued irritation of excessive perspiration. The disease is limited to those parts of the skin containing sebaceous follicles. His treatment consists in oily applications to the body-surface and the wearing of cotton next the skin. **Skin Diseases—** *continued*

Wellman[1] has described a severe chronic pemphigoid disease of West Africa associated with the presence of a diplococcus, and Clegg and Wherry,[2] dealing with the etiology of pemphigus contagiosus in the Tropics, summarise their findings and conclusions as follows:—

1. From cases of *Pemphigus neonatorum* and one case of *Pemphigus contagiosus* in an adult, micrococci similar to those described by Almquist were isolated.

2. Although occurring as well-defined kidney-shaped diplococci in the contents of the vesicles, the organism may, on superficial examination of cultures, be confounded with *Staphylococcus pyogenes aureus*. Our cultures did not produce indol in broth, and the diplococcus arrangement was reproduced in milk, or, better, in serum broth cultures.

3. A single human inoculation experiment with this organism produced typical but abortive vesicles. The essentially superficial nature of the inflammatory process set up in the human skin—resulting in the exudation of serum and leucocytes, and the formation of vesicles and the absence of any tendency to penetrate into the deeper tissues—certainly differentiate this micrococcus from the ordinary pyogenic cocci.

4. We believe it advisable to call the disease *Pemphigus contagiosus*, whether occurring in children or adults, and the etiological factor would then best be termed *Micrococcus pemphigi contagiosi*.

5. Cases of typical *Impetigo contagiosa* should be examined along similar lines, as the disease described under this name is possibly due to the same micro-organism.

Sleeping Sickness. Considering, in the first place, methods of spread, one finds that in the latest report[3] of the Liverpool expedition to Rhodesia, mention is made of work in Uganda, where successful transmission experiments were made with *Glossina fusca*. It is possible also that *G. morsitans*, *G. pallidipes* and *G. longipalpis* are also implicated, though in this connection one would quote Neave,[4] who in Northern Rhodesia found a place which had become infected both as regards *G. palpalis* and man from a locality 150 to 200 miles distant. Tracing the route of caravans back to this locality, he found the intervening country infested with *G. morsitans* but no *G. palpalis* and no sleeping sickness existed. Hence he thinks *G. morsitans* should be considered not guilty until the contrary is proved.

At present it would appear that not only tsetses but all biting-flies must be considered as possible carriers. Thus in the French Congo, Martin, Lebœuf and Rubaud[5] have noted how young children are often affected, and think that certain "domestic" insects, such as mosquitoes of the genera *Stegomyia* and *Mansonia*, may be to blame. This, however, requires confirmation. In Rhodesia all the work goes to show that the transmission is mechanical.

Koch,[6] as a result of work in Uganda and German East Africa, is of opinion that though it may be possible to infect *G. fusca* and *G. pallidipes* with the trypanosome, this must occur so rarely under natural conditions that they may be disregarded as conveyers of the trypanosomes. The same may be said of *G. morsitans*, which, he thinks, attacks man very exceptionally. This is contrary to the experience of most observers, and certainly, in the Bahr-El-Ghazal, *G. morsitans* is a pest to man and animals alike.

Koch also notes that though dogs and monkeys are known to have become naturally infected, the occurrence is so rare and the animals have died so quickly after infection that practically they may be disregarded as reservoirs of the disease.

He has further drawn attention to the probability of the disease being communicated by coitus. Thus, of 26 women in the German segregation camp, where there was a total of 425 cases, 7 had never been in sleeping sickness regions. It would seem that they

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[1] Wellman, F. C. (August 1st, 1907), "Description of a Diplococcus found in the lesions of a severe, chronic pemphigoid Disease in West Africa." *Journal of Tropical Medicine and Hygiene*, Vol. X.

[2] Clegg, M. E., and Wherry, W. B. (March 2nd, 1906), "The Etiology of Pemphigus Contagiosus in the Tropics." *Journal of Infectious Diseases*, Vol. III. Chicago.

[3] Reviewed in *Lancet*, April 11th, 1908, p. 1110, Vol. I. 1908.

[4] Neave, S. (April 25th, 1908), "Distribution of Glossina." *British Medical Journal*, Vol. I. 1908.

[5] Martin, G., Lebœuf and Rubaud (March 11th, 1908), "Epidémies de maladie du sommeil au Congo Français." *Bull. Soc. Path. Exot.*, Vol. I.

[6] Koch, R. (November 14th, 1907). *Deut. Med. Woch.*, p. 1889. Quoted in *Lancet*, 30th November, 1907 p. 1578.

Journal of the Royal Institute of Public Health, December, 1907, p. 751.

Journal of Tropical Medicine and Hygiene, February 15th, 1908, p. 68.

Sleeping Sickness— *continued*

had contracted the disease from their husbands, all of whom had died of sleeping sickness. When sleeping sickness was found in villages outside the glossina belt, women only were found to be infected, the children and men who had not visited sleeping sickness districts being unaffected. In one case the three wives of a man suffering from sleeping sickness contracted the disease. As has since been pointed out, if the converse can take place and males be infected by females, it is quite possible that the tsetse fly is only an occasional agent of transmission, and the outlook, therefore, very grave.

The above-mentioned French observers point out that Koch's views do not explain certain cases observed by them. They have never found the disease limited to married women, and, as stated, have often found young children affected. At the same time, they noted how the disease spreads amongst families and how the natives of the French Congo dread infection by contagion and take measures to isolate the sick.

The question as to whether or not the trypanosome passes a stage of its life cycle in *G. palpalis* still remains unsettled, despite the work of Minchin and his colleagues.

Koch lays stress on the fact that on two occasions parasites have been found in the salivary glands of the fly and on the presence of several forms of *T. gambiense* in its alimentary canal. He, therefore, believes that such a developmental cycle exists. How long a glossina can remain infective is not known, and this is a question which requires to be settled at an early date. Koch believes the fly to be long-lived, as it is not known to have any natural enemy, while its reproductive energy is feeble.

Minchin[1] found that the capability of infecting a vertebrate animal only lasts for a period of 48 hours; with a longer interval no infection was obtained. It was also noticed that freshly-caught flies may produce infection with trypanosomes without having been fed previously on infected animals. Experiments made upon monkeys show that many freshly-caught flies are either free from the infecting agent or that some monkeys are immune. Thus in one case 2299 flies were fed on a monkey over a long period without infecting it, whilst in another case as small a number as 134 flies produced an infection in it. When infection did take place it occurred much more rapidly with the freshly-caught flies than with those that had been fed upon animals infected with trypanosomes. In experiments made with flies fed on an infected animal, Minchin found that he could infect nine out of ten animals each with a single fly.

Hodges,[2] in his very valuable Uganda Report, deals with the source whence the fly may derive the trypanosome. He says:—

> Although vertebrates of various kinds have been artificially inoculated with *Trypanosoma gambiense*, yet, so far as is known at present, there is no wild or domestic animal which, itself almost or quite immune, carries this *Trypanosoma* so habitually as to act as a "reservoir" for the infection of sleeping sickness, as do the big game in the case of the *Trypanosoma brucei*, of Nagana. No animal, indeed, except the native dog, and that in only a few instances, in places where the degree of local infection has been intense and the epidemic has been of considerable duration, has been found to be naturally infected; on this point, however, further research is much needed, for, if there be such a reservoir, it is most likely a domestic animal, and might possibly be the native dog itself; at any rate, it is unlikely to be an animal which ranges, and would therefore carry the infection, very widely. It is obvious that animals which can become naturally infected by *Glossina palpalis*, unless the fact is very exceptional, are an added danger to the community.
>
> Meantime we must provisionally suppose that only the human being and the fly need be seriously considered as agents in the spread of infection, that the vast majority of flies, where human beings are scanty or absent, are uninfected and harmless in themselves, and that, if the fly could be eliminated from places of human concourse such as mentioned above, those existing elsewhere would run little chance of becoming infected.

He also suggests that it is even possible, still, that the conveyance of the infection by *Glossina palpalis*, rather than by other *Glossinæ*, or by any blood-sucking insects, may be merely owing to a prolonged viability of the trypanosome in the interior of this fly; that a migration rather than an evolution takes place, and that it is not a true host.

Manson[3] brings forward an interesting point in a recent paper, by saying that:—

> It is not a little remarkable that, of the ten cases of trypanosomiasis in Europeans which have come under my personal observation, three of them were females. Considering the very small number of European females and relatively large number of European males in tropical Africa, this large number of females attacked with trypanosomiasis is a striking circumstance. I am dealing, it is true, with very small numbers, and it is quite possible that the relative disproportion I remark on is accidental; but when we reflect that whereas women in Africa expose themselves, as compared to men, comparatively little to the conditions favouring the attack of *Glossina palpalis*, the disproportion becomes still more striking.

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[1] Minchin, E. A. (March, 1908). *Quarterly Journal of Microscopical Science.*

[2] Hodges, A. D. P. (December 22nd, 1908). Report of P.M.O. East Africa and Uganda Protectorates, p. 19.

[3] Manson, Sir P. (March 2nd, 1908), "My Experience of Trypanosomiasis in Europeans, and its Treatment by Atoxyl and other Drugs." *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. II., No. 1.

Sleeping Sickness—*continue*

Another point that has attracted my attention in connection with these cases is the frequency (four in the eight cases in which the point was inquired into) with which the symptoms were immediately ante-dated by what was described as a bite on the leg. The biting animal may have been a *Glossina*, but in the case of females—and two of the bitten ones were females—one would suppose that the petticoat would afford a protection even more effective than the trouser does in men.

Too much weight must not be attached to what may have been mere coincidence; but these facts are curious, and suggest further inquiry as to the possibility of some blood-sucker, perhaps some species of house vermin, being an occasional vector of *Trypanosoma gambiense*.

Next, as regards the disease itself and the parasite which presumably causes it, we find Martin and Darré[1] describing certain nervous symptoms observable at the onset of the malady. In one exceptional case, there was a general cutaneous hyperæsthesia save on the plantar and dorsal surface of the feet where anæsthesia was marked. There was also a partial paralysis of the extensors of the great toe. Babinski's sign was present. The symptoms slowly disappeared under treatment with atoxyl. The authors note that pains in the feet often constitute one of the first symptoms and that they are always very persistent. They lay great stress on this intense hyperæsthesia, which they say permits of an early diagnosis being made and treatment being commenced at once, when ultimate cure is more probable. They append a note by Kérandel on this symptom, which appears during the second month of the illness and becomes marked during the third month. The slightest contact with any hard object gives rise to acute pain, so that great care is exercised by the unfortunate patient in sitting or lying down, taking hold of objects, passing through a doorway, etc. The legs, the fore-arms and the hands are the parts most often affected. The pain is sometimes sufficiently severe to make the patient cry out. Although severe, it is very fleeting, passing off in from two to five minutes, and it disappears in a few days under atoxyl treatment. This hyperæsthetic condition is said to be pathognomonic, and it is suggested that it should be termed "signe de Kérandel."

Attention has recently been directed to the craving for meat displayed by sleeping sickness patients, and Mr. Archibald informs me that along with this a great desire for salt was evinced by patients in the Ugandese camps.

Moore and Breinl[2] have a very important paper on the morphology and life-cycle of *T. gambiense* which is likely to provoke much discussion. They quote Dutton, Todd and Hannington, who, dealing with the observations of Bruce and others in Uganda as to flies being non-infective after forty-eight hours, stated:—

We believe either (1) that something is wrong in the way in which *Glossina palpalis* has been used in these experiments; or, (2) that *Trypanosome gambiense* can be conveyed by some other means than by it.

Moore and Breinl say:—

So far, then, from its being established that sleeping sickness is normally spread among the African population by the bites of *Glossina palpalis* alone, it would seem that the most recent work on this subject indicates that possibly the infection through flies is in the nature of an accident, and that the means by which sleeping sickness spreads, in the manner in which it does spread in the African interior, has yet to be discovered.

They proceed to show that *T. gambiense* varies very much in size in the same blood, but they are unable to subscribe to the opinion that there are male, female and indifferent forms.

By special staining methods they demonstrate structures not hitherto described, such as an intra-nuclear centrosome, as distinct from the extra-nuclear centrosome or blepharoplast. There may, indeed, be several of the latter. They deal with the mode of multiplication of *T. gambiense* in the blood, and especially with the changes in the trypanosomes relative to the stage of infection. They observed a curve of infection, the number of parasites increasing and diminishing in the blood, and direct attention to the formation of what they call "latent bodies," found in the lungs, bone marrow and spleen. These latent bodies eventually become transformed into small trypanosomes, but apparently only a certain proportion of them undergo this change, the others disappearing. This indicates a complete cycle in the blood of a single host, the rat being the animal studied.

In rats the latent forms pass gradually into trypanosomes, these in turn divide through many generations, and their multiplication is followed by a metamorphosis which, whether we regard it as a special form of sexual process, as a form of pathogenesis, or as a sexual stage, the fuller details of which have not yet been

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[1] Martin, L., and Darré (January 22nd, 1908), "Sur les symptomes nerveux au début de la Maladie au sommeil." *Bull. Soc. Path. Exot.*, Vol. I.

[2] Salvin-Moore, J. E., and Breinl, A. (November 9th, 1907), "The Cytology of the Trypanosomes." *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. I., No. 3.

Sleeping Sickness— *continued*

elucidated, seems undoubtedly to stand in one of these relationships to the normal cell multiplications preceding the formation of latent bodies. The stage in question results in the production of the latent bodies once more, and the cycle is complete.

It may be objected to this conception that, notwithstanding the cyclic development of *Trypanosoma gambiense*, still there may exist a possibility, or probability, of the transference of the trypanosomes into some other host where a further metamorphosis, representing the sexual stage of the organisms, is passed through. This, of course, may be so, but we have in the case of the trypanosomes of *Dourine* a clear instance of a trypanosome life-history, which, under normal circumstances, is not transferred into any other kind of host; and, under normal circumstances, *Trypanosoma equiperdum* must pass through whatever sexual stage it may possess, its whole life-history in fact, in the body of the horse. *Dourine* can, however, like sleeping sickness, be inoculated from host to host by simple transmission of blood as well as by coitus; in other words, the faculty of being transmitted by simple inoculation of blood is shared by *Trypanosoma equiperdum*, wherein no other host is usually involved, as well as by *Trypanosoma gambiense*. In these circumstances, it is simply natural, assuming flies to be the agents by which sleeping sickness is transmitted, to admit that this form of transmission may be merely in the nature of a mechanical transference, and have no more relation to the sexual act in the life-cycle than has the artificial withdrawal of blood from a horse infected with *Dourine*. In other words, it would seem that the transference by flies in the case of sleeping sickness may have no more significance with respect to the life-history of the parasite than has the direct inoculation of *Dourine* from horse to horse by means of a needle.

As regards methods of diagnosis, a recent paper by Martin and Lebœuf[1] may be cited. They have compared the value of the different methods employed for making a microscopic diagnosis of *T. gambiense*. In all they examined 258 cases with a view to determining what is the procedure which enables one to discover the trypanosomes (i) with the maximum of certainty and (ii) in the minimum of time, and that which natives will submit themselves to with the least outcry. They recommend :—

1. Examination for 10 minutes systemically (a movable stage being employed) of a fresh film of blood taken from the pulp of the finger, or preferably two such films taken from two different fingers, the one from the right hand and the other from the left hand.

2. If this first examination is negative, and if the patient has enlarged glands, these should be punctured in the cervical region for choice. If the neck glands are too small, try the sub-maxillary or inguinal.

3. If the parasites can still not be found, take 10 c.c. of blood, centrifuge and examine the leucocyte layer, making, if necessary, a couple of films.

4. If the result be still negative, the case is probably healthy; but, if necessary, and there is no objection, proceed to lumbar puncture, removing 10 c.c. of the cerebro-spinal fluid and centrifuging it for 15 minutes.

Nattan-Larrier and Tanon[2]* advise scarification of the erythematous patches for detection of the trypanosomes, which may be found in this way when absent from the blood. This refers specially to white-skinned patients.

Finally, the all important questions of prevention and treatment may be considered together.

A portion of the summary of Hodge's Uganda Report[3] may be quoted with advantage. He says :—

(a) The distribution of sleeping sickness, which was pretty well known before, except in the case of the Nile Province and the small epidemic near Elgon, has been confirmed, and the observations appear to connect the disease more closely than ever with *Glossina palpalis*.

(b) Broadly speaking, the degree of infection and the distance of penetration (other things being equal) into the hinterland is everywhere proportionate to the intensity of the infection and the prevalence of fly at the corresponding lake-shore or river-side.

(c) The enquiry has shown the limited extent of the "infective areas," in which alone sleeping sickness is communicable to man, and the wide extent of the fly-free interior, in which it is not communicable.

(d) The "infective areas" form a very small proportion of the epidemic areas, and the bulk of human infections is due to communication with these areas, while only a small minority is caused by actual residence within them.

(e) Investigation shows also the efficacy of clearing, when scientifically applied, the apparent feasibility of segregation, and the importance of obtaining native co-operation, if possible.

(f) By clearing or otherwise destroying the narrow "natural" range of *Glossina palpalis*, the wider "following" range is abolished.

(The narrow "natural" range appears to be about from 30 to 100 yards in width, being the strip of scrub and undergrowth near water haunted by "the fly").

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[1] Martin, G., and Lebœuf (February 12th, 1908), "Diagnostic Microscopic de la Trypanosomiase humaine." *Bull. Soc. Path. Exot.*, Vol. I.

[2] Nattan-Larrier, L., and Tanon (October 17th, 1906). *Presse Médicale.*

[3] *Loc. cit.*

* Article not consulted in the original.

Sleeping Sickness—*continued*

(g) It is most important to consider, with regard to prevention, the width of the fly-ranges (infective areas) and the constant traffic with these from inland.

(h) The most important and most practical preventive measures at the present time appear to be a combination of the clearing and of segregation from the infective areas, with or without deportation. Also the segregation in fly-free country will favour the administration of any special treatment.

(i) Our action in the Nile Province, the Nile itself not being a true intertribal boundary, and there being constant migration from bank to bank, must depend on the result of our enquiries into the capacity of *Glossina morsitans* and *G. pallidipes* of carrying the infection, and also the action (if any) which may be taken by the Sudan, and, especially, the Congo Free State Governments in the matter.

(j) The natives of the Uganda Protectorate, by keeping their sick from the water side, their dwellings outside the fly-range, and their water supplies, fords, ferries, landing, markets, etc., cleared of undergrowth; placing them, wherever possible, in fly-free situations could, in all probability, themselves control the disease; and though it is not likely that the bulk of them will yet attempt it, it is possible that they may in the course of years gradually acquire a habit of using the defensive measures now proposed.

(k) It is probable that sleeping sickness may remain endemic in certain parts of the Protectorate which will become localised as time goes on. Whether the lake-shore or Nile-bank regions will remain permanently dangerous to a population living in them will depend chiefly on the natives themselves.

(l) It is most important that the duration of infection in the fly should be determined.

(m) In the Uganda Protectorate, although it may be impossible to eradicate sleeping sickness in a few endemic centres, I believe there is good ground for hope that the present epidemic may be so far controlled, over the greater part of its extent, that the disease, even though we fail to find effectual and practicable cure, may cease to be a menace to the population and a serious obstacle to the development of the country, and, further, that fresh outbreaks of anything like the dimensions of the present one should become almost impossible of recurrence in the future.

As regards the action of the Sudan, to which reference has been made, this, so far as the Nile is concerned, will be found detailed in the Second Report of these laboratories. It will be remembered also that all recruiting from Uganda was stopped.

Koch has made the following recommendations :—

1. Sick natives should be prevented from crossing the frontier.

2. A native found to be infected, no matter in what country or from what country, should be detained where he was infected.

3. Each country should interdict its natives from entering infected districts.

4. Segregation camps should be established.

5. Attempts should be made to destroy crocodiles, which Koch believes are the principal food-source of the fly. The eggs of the saurians should be destroyed and the adults poisoned.

6. Scrub and undergrowth should be cleared whether they constitute fly-belts or may afford dangerous shelter for flies, *i.e.* round posts and villages with water in their vicinities.

Some of these recommendations have been severely criticised,[1] and, although excellent in theory, would seem to be difficult if not impossible of achievement, considering the conditions which obtain in the countries infected.

Koch thinks the crocodile essential to the fly and the determining cause of its presence, because both abound in the regions of Lakes Tanganyika and Nyanza, while in Lake Kivu there are no crocodiles and *G. palpalis* is absent. Hodges, however, has shown that, in Lake Albert Edward, crocodiles are absent and *G. palpalis* abounds, while south of Gondokoro the reverse is the case. He thinks the fly may feed on the hippopotamus at times. Most of those to whom one has spoken on the subject seem to think that the tsetse will take blood from any available source, naturally preferring that which is most convenient and yields an abundant supply.

Cook[2] has stated that—

From the point of view of prophylaxis four important methods were being adopted in Uganda. First, those actually suffering from sleeping sickness were being collected so far as possible and segregated in isolation camps, where each man, the presence of trypanosomes having been verified in his body, underwent thorough treatment with atoxyl. Every care was taken to remove prejudices from the native mind, and an excellent work was being done. The second method was the compulsory eviction or removal of all natives from infected tsetse-fly areas. That bristled with difficulties. The Uganda police were too few in number to patrol a large area efficiently. The natives clung to their old homes and plantations and not one in a thousand believed that the tsetse fly, which they call "Kivu," really conveyed the disease. Moreover, the islands, the worst foci of the disease, were almost necessarily left alone. The third method was the clearing of extensive areas round landing stages, markets on the lake shore, watering places, and the necessary Government, or trading, stations on the lake. The fourth method was the printing in the native tongue of clear instructions as to

[1] Leader in the *Journal of Tropical Medicine and Hygiene*, February 15th, 1908, p. 55.

[2] Cook, A. R. (October 26th, 1907), "Report of Meeting of Society of Tropical Medicine and Hygiene." *Lancet*, Vol. II.

Sleeping Sickness— *continued*

the nature of the disease, how it was spread, the importance of clearing away the undergrowth in which the "Kivu" lived, and the urgent need for segregation of those actually sick, while they enlisted by every means in their power the active co-operation of the chiefs. In regard to personal prophylaxis, the European, even when living within the dangerous area, was but little exposed to infection if he used his knowledge intelligently. His bungalow should not be built near the water's edge or close to the native compounds. The ground for a considerable area round his station should be cleared and, what was more difficult, kept clear of undergrowth, especially of bushes, in the shade of which the tsetse fly was found. His dwelling should be protected by gauze screens or fine wire netting and his bed by a mosquito net, while the native porters who brought his water from the lake should not be allowed to enter the house, for tsetse flies often buzzed persistently round the heads and shoulders of those porters and followed them for long distances. Tsetse flies had been discovered inside European bath-rooms at Entebbe at some considerable distance from the lake—doubtless introduced in that way. Koch told him that he and the officers of his expedition were simply depending on living away from the water's edge, which the tsetse fly frequented—their camp was some 400 feet above lake level—and on wearing white garments, since the fly was repelled by white but attracted by dark clothing.

As regards the threatened portion of the Sudan, the work carried on by Major Dansey Browning and Captain Ensor has furnished a great deal of information. A consideration of the latter officer's lengthy and able report has been undertaken by Bimbashi Archibald, who, in view of his experience in Uganda, is well qualified to comment upon the various points discussed (Third Report).

At the present moment, and from a practical standpoint, treatment may fairly well be summed up in the word "atoxyl."

Koch's method of administration is to give half a gramme of the drug by subcutaneous injection on two successive days, and after ten days to repeat the injections. In 20 per cent. of cases so treated the trypanosomes disappeared permanently, but in other instances they returned. Hence it is necessary to repeat the treatment every ten days during two months. It is then suspended for an indefinite time and, on any return of symptoms, repeated, if necessary, every two months. Recent observations, however, have shown that cases supposed to be permanently cured do show relapses, and this without any possibility of re-infection from tsetse fly (Archibald).

The disappearance of trypanosomes from the blood after atoxyl injections is attributed by Koch to a state of immunity produced by an absorption of dead parasites.

Atoxyl is very effective in diminishing the size of the enlarged lymph glands, but one of its drawbacks is that it is apt to produce permanent blindness if given in excessive dosage.

Hence acetylated atoxyl has been introduced and used by Ehrlich, Nierenstein[1] and others. It is an antipyrine containing arsenious acid. Mention may also be made of the Liverpool method of using atoxyl combined with corrosive sublimate[2] or with orpiment, as tried by Laveran and Thiroux,[3] who have obtained good results in animal experiments by this procedure.

Antimony salts were introduced by Plimmer and Thomson,[4] and stated to be better than atoxyl, but latterly less has been heard of this method.

Other remedial measures and accounts of experimental work carried on in the Sudan will be found in the special article on "Trypanosomiasis" (Third Report),[5] but papers of value are those by Boyce and Breinl[6] and by Manson.[7] The latter's conclusions of his results, as regards the treatment of trypanosomiasis in Europeans by atoxyl and other drugs, are here quoted:—

1. Trypanosomiasis in man is not necessarily a fatal disease.

2. Atoxyl has a marked effect in checking the clinical manifestations of the infection, and in causing the parasites to disappear from the peripheral circulation.

3. Notwithstanding continuation of atoxyl treatment, parasites may reappear again and again at uncertain intervals, and usually concurrently with a rise in temperature.

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[1] Nierenstein, A. (July 27th, 1907), "The Treatment of Trypanosomiasis." *Lancet*, Vol. II.

[2] Moore, Benj., Nierenstein, M., and Todd, J. L. (February 1st, 1907), "A Note on the Therapeutics of Trypanosomiasis." *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. I., No. 1.

[3] Laveran, A., and Thiroux, A. (February 25th, 1908), "Recherches sur le traitement des Trypanosomiasis." *Ann. de l'Institut Pasteur*, Vol. XXII.

[4] Plimmer and Thomson (November 7th, 1907), "Further Results of the Experimental Treatment of Trypanosomiasis." *Proceedings of the Royal Society.*

[5] Much curtailed owing to the losses sustained by fire.—A. B.

[6] Boyce, Sir R., and Breinl, A. (March 2nd, 1908), "Atoxyl and Trypanosomiasis." *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. II., No. 1.

[7] *Ibid.*

Sleeping Sickness—*continued*

4. Nevertheless, if the drug be persevered with, the parasites ultimately disappear for good and do not return.

5. Large doses of atoxyl are not necessary to secure this result.

6. Large doses of atoxyl should be avoided, as they are apt to cause serious lesions, peripheral neuritis, optic atrophy, gastro-intestinal inflammation, and other toxic conditions which necessitate suspension of a valuable remedy.

7. Trypanroth, mercury and parafuchsin seem ineffective in human trypanosomiasis.

8. Antimony may have a therapeutic influence in trypanosomiasis, but the hypodermic injection of the sodio-tartrate is impracticable.

He considers the prospects of atoxyl treatment as being most hopeful, and suggests for the routine treatment of trypanosomiasis a two to three grain dose of atoxyl every second or third day and kept up for at least two years. Concurrent disease, such as malaria, syphilis, etc., should be treated at the same time, and the patient should return to his native country, be spared fatigue, worry, exposure and excess of all kinds, and be placed under the most favourable hygienic surroundings.

One may complete this review of Sleeping Sickness by tabulating the lines along which further research should be conducted as formulated by Koch :—

(*a*) The discovery of a method of artificially infecting *Glossina palpalis*.

(*b*) The comparative study of the trypanosoma found in *Glossina palpalis* in non-sleeping sickness areas with the trypanosoma found in sleeping sickness areas.

(*c*) The determination of the proportion of naturally-infected *Glossina palpalis*.

(*d*) The determination of the period for which trypanosoma infection persists in *Glossina palpalis*.

(*e*) The use of the complemental method for the diagnosis of trypanosomiasis.

(*f*) Experiments with new remedies.

(*g*) Further investigation of the natural food supplies of the fly.

(*h*) The study of the distribution of the fly on rivers and the conditions determining their presence.

(*i*) Experiments for the purpose of infecting glossina reared from larvæ, and of transmitting infection by a fly reared in this manner.

Mesnil[1] has recently reviewed the work of the members of the French Mission for the study of the disease in the French Congo. The following points may be mentioned :—

1. As regards the value to be attributed to glandular enlargement the French observers differ from Dutton and Todd. They find that enlargement of the lymph glands is not synonymous with trypanosomiasis. Many individuals exhibiting hypertrophied cervical glands do not suffer from the disease, and, conversely, patients with trypanosomiasis have not always marked adenitis.

2. He quotes the work of Martin and Lebœuf, already recorded, as regards the microscopic diagnosis of trypanosomiasis.

3. The rapidity which which Europeans are attacked on the Congo is insisted upon.

4. Atoxyl alone is not sufficient to effect a cure. Picric acid has been given along with it internally, and this method seems promising.

5. Proof of the rôle of *Glossina palpalis* as carrier is to be found in the geographical distribution of the fly and the disease, and the absence of the latter in the Antilles, into which it must have been repeatedly introduced in the old slavery days.

6. It was noticed, however, in the French Congo that the number of cases of sleeping sickness in no way corresponded to the number of *Glossina palpalis* present in the district. Thus, in places where tsetse flies abounded, cases were no more numerous than in hilly districts, where there was little water and flies were few.

7. Family infections were noted, but Koch's suggestion as regards spread by sexual intercourse cannot explain the infection in children, and it is thought that mosquitoes of the genera Mansonia and Stegomyia, especially the latter, may act as carriers of *T. gambiense*.

8. Roubaud has found that trypanosomes sucked up by a tsetse fly along with the blood of an infected mammal are arrested in their passage along the insect's proboscis, and that they undergo modification and evolution in its saliva with extreme rapidity. They become firmly fixed to the wall of the proboscis by the extremities of their flagella, their undulating membranes completely vanish and their centrosomes change position, becoming anterior to their nuclei. In short, they assume *Herpetomonas* forms. In this position they multiply very abundantly during the first hour following the insuction of the blood. Their duration of life in the proboscis of the tsetse does not seem to be more than two days in the case of *T. brucei*, but for *T. dimorphon* it is four and a half days, and for *T. gambiense* five days.

This is regarded as a specific development, and it occurs only in tsetses which have imbibed infected blood. Naturally this important observation requires confirmation, but it is regarded as upholding the views of Bruce enunciated in 1904.

These, then, are the main points in an interesting review of interesting and very suggestive work.

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[1] Mesnil, M. (February, 1908), "Documents Français sur la Maladie du Sommeil." Extrait., *Assoc. Scientif. Internat. D'Agronom. Coloniale.*

Small-pox. De Korté[1] gives the following technique for demonstrating the organisms found by Funk and himself in variolous matter and in human vaccine lymph, and which he named provisionally *Amœba variolæ vel vacciniæ*:—

To be satisfactorily seen, a hanging-drop preparation of variolous or vaccine lymphs must be made. Avoid all manipulations of whatsoever kind. All ordinary methods of staining, fixing, or drying, the pressure of the cover slip and desiccation invariably lead to rupture of the ectosarc, and to disintegration of the parasites; for these reasons, in smear preparations, only the detritus of the parasites is to be found. To obtain sterile variolous and vaccine lymphs, wash the pock with an antiseptic without rupturing its walls, remove any excess of disinfectant, and force a sterile capillary tube into the interior of the vesicle; by depressing the free end of the tube the vesicular contents will gravitate into the tube. For storing purposes the ends must be carefully sealed in a flame. The lymph being thus collected, in the case of human vaccinia on the ninth day of eruption, centrifugalise the tube to concentrate the parasites, as there are comparatively few amœbæ present. From the centrifugal end of such a tube allow a small drop to exude on to a cover-slip, and put up as a hanging-drop on a warm stage. At 98° F. the amœba puts out pseudopodia and is actively motile. The spores, being excessively minute and not very refractile, can be seen, but only with difficulty, at the edge of the specimen. Small-pox matter should be collected about the fifth day of eruption or earlier, and similarly treated, but it is unnecessary to centrifugalise, as the parasites are present in enormous numbers. In glycerinated calf lymph the parasites and spores are best seen suspended in a drop of normal saline solution, the method of procedure being identical in other respects with that indicated for variolous matter. All these parasites can be stained *in vivo* by suspending them in a normal saline solution tinted with aqueous safranin, Lœffler's blue or Bismarck brown. The following method of making permanent preparations, unsatisfactory though it is, may be partially successful when absolutely fresh material is procurable; after keeping *in vitro* the parasites will not admit of this procedure. Spread on a cover-slip a small drop of variolous or human vaccine lymph (that from a monkey is more satisfactory) with the edge of a second cover-slip, avoiding pressure as far as possible; immerse immediately in equal parts of spt. rect. and ether for ten minutes, and then stain with Lœffler's blue for five minutes. The parasite of variola stains irregularly, the nucleus probably not at all. Mount in Canada balsam. Glycerinated calf lymph spread in a thin film in the above manner can be fixed with the fumes of 2 per cent. osmic acid for five minutes, stained with dilute aqueous safranin for ten minutes; it can then be mounted permanently in equal parts of glycerin and water in a shallow cell, thus avoiding the pressure of the cover-slip, which would destroy the organism. Permanent preparations are, however, distinctly unsatisfactory, but anyone taking the trouble to follow the directions laid down will not have the least difficulty in seeing the *Amœba variolæ vel vacciniæ* in a hanging-drop preparation.

It is an amœboid protozöon measuring about 1/2500 of an inch in diameter, having the form of an oblate spheroid, containing intra-cellular spores, and in the case of the parasite found in human vaccine lymph, actively amœboid. In human vaccine lymph it is to be found on or about the ninth day of vaccination, after which it disappears spontaneously.

In a later paper[2] he describes the organism more fully, saying:—

Let a smear be made on a cover-glass of some variolous lymph at the seventy-second hour of the eruption; let the preparation be fixed with heat or other fixative (except Leishman's or Jenner's stain) and stained with the ordinary dyes. The specimen will present a granular detritus with few, if any, cellular structures. Take, however, some of the lymph from the same vesicle, and make a hanging-drop preparation. The latter will now present a very different appearance. It will be seen to contain a large number of morphological elements resembling pus cells, containing highly refractile grains within the cell substance. Among them will be seen large cells having a definite nucleus and karyosome, and limited by a thick-walled ectosarc; this form I have termed the encysted parasite. Again, as more readily obtainable, fix and stain a cover-slip preparation of fresh active glycerinated calf lymph; beyond some epidermic cells, nothing but a granular detritus is visible on examination. Yet this same lymph suspended in normal saline solution, will display, in addition to the refringent grains constituting the granular detritus, a large number of circular elements varying in size, some having thick ectosarcs, also some less numerous segmented bodies; these may consist of a few or of a considerable number of segments; this body I have termed the morula, and it probably represents amitotic multiplication of the germs.

If the vesicular contents of a human vaccine vesicle be examined as a hanging-drop with the highest powers of the microscope and suitable adjustment of the light, disregarding any gross cell element which may be present, the fluid will be seen to contain a vast number of exceedingly small grains, which appear to be motile on a warmed stage; their shape and size, because of their smallness, is practically indeterminable. These grains, I think, are spores; they are larger and more distinct in variolous matter and always normally situated within the cell; they are motile in calf lymph, immobile in glycerinated calf lymph. The various bodies thus far described will be seen to consist of four elements—spore, sporidium or amœba, encysted parasite, and morula body found in glycerinated calf lymph. Two objections have been raised to the parasitic nature of these bodies. It has been affirmed on the one hand that they are degenerate epithelial cells, and on the other hand that they are nothing but leucocytes. Both arguments are refuted by the experiments already described. There is no difficulty in fixing epithelial cells or leucocytes. An object cannot both be present and absent from one situation at one time, yet at a period when all are agreed on the absence of leucocytes from the vesicular contents of the pock, the bodies above described are seen to be present in large numbers in hanging-drop preparations. If the variolous matter be gathered with antiseptic precautions and stored in a capillary tube, at the end of twelve months the parasites will be found to be present provided they be sought for in a hanging-drop preparation, not otherwise. It is extremely improbable, that leucocytes will remain intact for this period outside the living body in whatsoever manner they may be kept. It may be objected that variolous lymph has a specific conservative action on leucocytes, and that they may live in this fluid for a very long time. For this assumption there is no warrant; for if

[1] De Korté, W. E. (November 19th, 1904), "The Parasites of Small-pox and Vaccinia," in Report of Meeting of the Pathological Society. *British Medical Journal*, Vol. II.

[2] De Korté, W. E. (December 1st, 1906), "The Virus of Small-pox and Vaccinia." *British Medical Journal*, Vol. II.

human vaccine lymph be gathered on the eighth or ninth day of the eruption in a capillary tube and subsequently centrifugalised—if the centrifugilate be examined as a hanging-drop, certain cellular elements very like leucocytes will be seen; if the preparation be kept for seventy-two hours longer the cellular elements will have disappeared. It can therefore be affirmed that in human vaccine lymph the bodies which are assumed to be leucocytes disappear in a comparatively short space of time. Furthermore, the parasites in the forms of spores, encysted parasites, and morulæ have no morphological resemblance whatever to either leucocyte, lymphocyte, or epithelial cell.

In the same article he points out that the evidence is so far against a bacterial cause for small-pox and vaccinia, and suggests that in small-pox the secondary fever is due to the pyogenic organisms which gain access to the pock about the seventh or eighth day. Moreover, he refers to the fact of the complete absence of infectivity during the incubation period of the disease, and, as additional evidence of the nidus of the virus being the skin (in which it is imprisoned during the incubation period), cites a case where variola was conveyed during the incubation period, to a second individual, by means of skin grafts.

He alludes to the organisms described by Guarineri and certain American observers, stating that it is impossible to say whether these are different from those described by Funk and himself or whether they represent another stage in the life-history of the same organism. He and Funk deal with the contents of a lesion which is practically extra-corporeal, the others with an intra-corporeal lesion.

One cannot deal at length with the parasite of Guarnieri, *Cytoryctes variolæ*, which has been worked at by Calkins, Councilman, Bancroft and others. The technique required for its demonstration is more complicated necessitating the preparation of skin sections. Councilman[1]* states:—

It is believed that the organism which constitutes the virus of vaccinia and small-pox is the same: that in vaccinia it undergoes a definite cycle of development, resulting in a structure, the gemmules arising from simple growth and segmentation; that in small-pox a further and more complicated cycle of development, in which probable sexual forms occur, is added to the vaccine cycle. It is only in man and in the monkey that the conditions are favourable to the development of the cycle which constitutes small-pox. The intranuclear parasites are as characteristic for small-pox as are the cytoplasmic forms for vaccinia. They are found in both variola inoculata and in variola vera. The spores which arise from the multiplication of the intranuclear bodies constitute the contagion of small-pox, which is capable of air transmission. This introduced into a susceptible animal develops the typic disease, small-pox, both cycles of the organism taking place in the lesions. In the non-susceptible animal, such as the calf or rabbit, only the single, and probably a sexual, cycle is developed constituting vaccine.

Reference may also be made to a long extract in the *Medical Annual* for 1907. Davies[2] epitomises the subject as follows:—

In every sporozoon definite reproductive phases occur, characteristic of some phase of disease, or of some peculiar environment of the parasite, as, *e.g.* in the malarial parasite.

Stage A.—Asexual reproduction or schizogony (takes place in the blood corpuscle of man or bird).

Stage B.—Sexual cycle or sporogony (takes place in the digestive tract of the mosquito).

Apparently, from Calkin's observations, an analogous series of processes takes place in the parasite causing vaccinia and variola; cytoryctes variolæ, with this important variation:—

Stage A.—Asexual phase or schizogony (vaccine body) (takes place within the cells of the rete mucosum outside the nucleus).

Stage B.—Sexual cycle (takes place within the nuclei of these cells).

Thus the complete cycle of multiplication and of sexual reproduction is perfected in one host.

(*a*) The first development of the germ in the host is unknown; probably a multiplicative reproduction occurs, as a result of which gemmules are carried by the blood to the skin, where the further development takes place; so much is conjectural. From this point observations are fairly complete.

(*b*) The gemmules become intracellular (cytoplasmic) amœboid organisms, which give rise to similar gemmules (Councilman's vaccine cycle). This process must continue for some time, as the gemmules are, in variola, distributed to all regions of the skin.

(*c*) Ultimately the germs derived in this way give rise to forms which penetrate the nuclear membrane and develop into gametocytes of two types, male and female. Conjugation probably follows, zygotes develop into a large amœboid organism in which pansporoblasts originate, which give rise to primary sporoblasts, and these to multitudinous spores; all this propagative reproduction takes place within the nucleus.

(*d*) The infection of fresh nuclei; and

(*e*) The transmission to new hosts, may be readily grasped.

An important point is that evidence of every stage in the first (cytoplasmic cycle) indicates that in vaccinia and variola we have to do with the same organism which in vaccinia has undergone some modification by reason of which the nuclear phase is inhibited.

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[1] Councilman, W. T. (October 21st, 1905). *American Medicine.*

[2] Davies, D. S. (March, 1907), "Diphtheria and Small-pox: an Epidemiological Contrast." *Public Health*, Vol. XIX.

* Article not consulted in the original.

Small-pox *—continued*

In this same paper the author notes that we have learnt of late years the preponderating importance of direct personal infection in the spread of most of the common, communicable disorders, and that in small-pox this becomes the simple factor which has to be controlled in order to suppress an epidemic outbreak. He states that in Bristol no quarantine beds are kept for contacts. They are not necessary, as contacts are not infectious till they sicken, and a careful system of visiting on the calculated day for sickening has always permitted all such cases to be headed off into hospital before further infection results. Work is not usually interfered with in the case of contacts who consent to immediate vaccination. Disinfection is attended to in the usual way, but the prime sources of infection in general are men not fomites. The control of small-pox, then, from introduced centres resolves itself into a careful personal search for contacts and the use of experience and judgment in looking for them in the right place.

This leads one to the vexed question of the ærial convection of small-pox, a subject which, so far as one knows, has not yet been definitely settled, despite the lengthy discussion of which it formed the subject, and which will be found fully detailed in the *Proceedings of the Epidemiological Society* for 1904-5 (*pages* 174-258).

Of interest in this connection is a paper by Vaughan,[1] on the Incidence of Small-pox in Calcutta, wherein it is stated that the small-pox hospital appeared to be a very small factor as compared with other influences favouring the spread of the disease, and certainly, as far as the native population is concerned, its influence was that of a drop in a bucket. One saw something of a small epidemic of variola in Omdurman some years ago. The cases were treated in tents situated at no great distance from a fairly populous neighbourhood, at least in one direction, and certainly there was nothing to show that the disease was spread by ærial convection.

A very valuable paper containing much that is not found in text-books is that by Thomas,[2] which one is tempted to reproduce in full. Lack of space forbids such wholesale pilfering, but certain points must be noted in detail. The incubation period is given as being generally from ten to twelve days, oftenest twelve, but it may be as short as six days and as long as twenty. After giving the signs and symptoms of the invasion period, he says :—

During the invasion stage, and before the appearance of the prodromal rashes, the diagnosis has to be made from :—

1. Other infectious diseases having an acute onset, *e.g.* measles, scarlatina, typhus, influenza, and depends primarily upon *(a)* Presence of an epidemic; *(b)* History of exposure with the appropriate incubation period, *(a)* and *(b)* in all cases.

In the case of the diseases indicated below, the following points should be considered :—

Scarlatina.—With rash absent or missed.—Condition of tongue, cervical lymph glands, tonsils, nose discharge, injection of soft palate (enanthem), circum-oral pallor, history of vomiting and sore throat. Backache absent or slight.

Measles.—Coryza, photophobia, lachrymation, Koplik's spots. Backache absent or slight.

Small-pox.—Headache and backache intense and unremitting. Vomiting may be present.

Typhus.—Backache not very pronounced. Headache intense, and very often associated with painful and tender eyeballs. Fæces characteristic, face rather dark red, conjunctivæ injected, eyes look heavy, expression dull and apathetic. Great and early muscular weakness. Vomiting uncommon.

Enteric Fever. Although this has not an acute onset, many cases are, when small-pox is rife, notified as small-pox. Attention should be paid to *(a)* Gradual rise of temperature at onset-step ascent on chart; *(b)* Early epistaxis or deafness not uncommon; *(c)* Widal reaction, this may be absent; *(d)* Tympanites; *(e)* Condition of tongue, spleen, stools.

Chicken-Pox. Complete absence of prodromal illness, save in adults, when this stage may be moderately severe. Rise of temperature, if present, and the appearance of the rash almost simultaneous.

Influenza. Here the diagnosis may be impossible until the time interval for the appearance of the rash has passed. The muscular soreness and prostration are both generally much more exalted in influenza than in small-pox. The history of exposure and the presence of an epidemic are of special importance here. The bacillus may sometimes be isolated from the sputum.

Meningitis. The history, with the presence of a possible cause, *e.g.* suppuration of the middle ear, or tuberculous focus in a lung, is important. The subsequent course, with the attending palsies, generally soon clears up the issue. Backache is uncommon.

Cerebro-spinal Meningitis. Retraction of the head. Rigidity of the neck muscles. Kernig's sign. Possible presence of the bacillus in the nasal discharge or in the fluid obtained by lumbar puncture.

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[1] Vaughan, J. C. (July, 1907), "On the Incidence of Small-pox in Calcutta." *Indian Medical Gazette*, Vol. XLII.

[2] Thomas, A. E. (January, 1908), "The Diagnosis of Small-pox." *Public Health*, Vol. XX.

Small-pox *—continued*

The initial rashes are then considered, morbilliform, scarlatiniform and hæmorrhagic types receiving mention and their diagnosis from those of measles (especially the papular forms), scarlet fever, and from septic rashes considered. The occurrence of small-pox without a rash is also mentioned. After the appearance of the rash which is described at all its stages, the diagnosis has to be made from :—

In all stages; chicken-pox, acne, syphilis, drug eruptions, glanders, scabies, lupus, especially of the face.

In the papular stage: prodormal rash of measles, erythema nodosum, lichen planus.

In the vesicular and pustular stages: herpes, erythema iris, and erythema bullosum.

In the pustular stage: impetigo, and pustular scarlet fever.

As regards chicken-pox, he notes that there is a type, chiefly found in adults, in which the face distribution may be nearly, if not equally, as intense as that on the trunk. The points distinguishing varicella from variola have been tabulated under the heading chicken-pox, and Thomas, after dealing with this question, points out that the greater depth of the initial skin lesion in small-pox explains :—

(1) the shotty character of the rash; (2) the pearly-yellow contents of the vesicle, the colour being due to the thicker epithelial covering; (3) the hardness and hemispherical surface of the vesicle; (4) the absence of the crenated edge in the vesicle. This is possibly damped out by the thicker layer of epithelium, just as the several layers of an onion hide the irregularities at the core; (5) the absence of early cupped scabs owing to the difficulty of rupture; (6) the pitting; (7) the thickness of the crusts; (8) the presence of "seeds" in the palms and soles; (9) possibly the umbilication and the formation of septa.

The superficial position of the lesion in chicken-pox explains: (1) the moderately soft character of the rash; (2) the clear transparent, almost colourless, contents of the vesicle, due to the very thin epithelial covering; (3) the soft and sometimes spherical or ellipsoidal surface of the vesicle; (4) presence of crenation or puckering in the vesicle; (5) early cupped scabs; (6) the absence of pitting, save in severe cases; (7) the thinness of the crusts; (8) the absence of "seeds" in palms and soles.

He notes that there is no one characteristic sign on which absolute reliance can be placed, and that it is often very difficult to distinguish moderately severe chicken-pox from mild small-pox. The rule is to consider whether the affection is trivial or grave. In the latter, vaccinate and treat as small-pox. In the former, vaccinate also if doubt persist and treat as chicken-pox.

In the Sudan the diagnosis from syphilis may give rise to trouble, and the following points will serve to distinguish the two diseases :—

History of exposure may be obtained in one or the other, and in syphilis the original chancre, its scar, or the usual secondaries may be recognised. In the male, where there is no chancre or its scar, the urethra should be examined for its presence.

Mode of Onset.—In syphilis, slow, insidious, the fever is not high, nor are the constitutional signs urgent or severe. There is no initial chill, no backache; the headache, if present, is not severe. The patient is able to go about his daily work; he does not lie up. The temperature does not remit with the appearance of the rash—there is no feeling of *bein aise*. In small-pox there is a sudden onset by chill, early high temperature, severe backache and headache, often vomiting. The patient lies up at home and stops work.

Rash.—In syphilis this takes many days to appear; in small-pox twenty-four to forty-eight hours. In syphilis there is no remission of temperature, no establishment of *bien aise*. The distribution of the syphilitic rash may be like, or unlike, that of small-pox. It is generally more copious on the trunk than on the face, and is rarely found in the soles and feet. The rash of syphilis is polymorphic, and may exist as papule pustules small and large, or vesicle concurrently. The pustules and vesicles of syphilis are usually conical, with deep subjacent ulceration; they are not flattened hemispheres as in small-pox.

Progress.—The regular sequence from papule to vesicle to pustule, with the proper time intervals, is present in small-pox, absent in syphilis. In the latter the development of the lesions is most irregular and slow.

Thomas asks, "Is Vaccination of any value as an aid to diagnosis"? and replies in the negative, going on to remark :—

There are rare cases on record in which patients efficiently vaccinated have subsequently passed through undoubted attacks of modified small-pox within a few months.

The possible consequences of even one unrecognised case of small-pox set free are so appalling that any uncertain criterion must be ruthlessly discarded. On the other hand, I have never seen a case of small-pox which could be successfully vaccinated within two years of the attack. We want to know the interval between a case of small-pox and the possible subsequent successful vaccination. Second attacks of small-pox are known, so that it is quite legitimate to assume that small-pox patients may be at some later period successfully vaccinated.

It has been stated that if vaccination be performed within three or even four days of exposure to small-pox, the threatened attack will in all probability be aborted. More definite information is required too on this head, so that the possibility of successful vaccination may

Small-pox —*continued*

become an efficient help in diagnosis. It is not at all uncommon in small-pox hospitals to see small-pox and vaccinia run parallel courses simultaneously in the same patient.

Finally, he tabulates sources of error in diagnosis as follows :—

1. Inaccurate history, *e.g.* former alleged attacks of small-pox. Too short or too long a period intervening since exposure.

2. Relying too much on the presence of vaccination scars, even when performed a few months previously. Their presence does not justify the exclusion of small-pox.

3. The formation of septa in the vesicle, estimated by pricking with a needle along the periphery. In small-pox the vesicles are said to stand, but in chicken-pox to collapse, being unilocular in the latter. This is a most unsatisfactory criterion and quite unreliable.

4. Presence or absence of umbilication—this, too, is no sure guide.

5. Being satisfied with the existence of a cause sufficient to explain the existing clinical complex without making sure that the cause thus presumably ascertained is the actual and effective agent—the *causa causans*.

In order to avoid this, it may become necessary in cases of difficulty to examine the various systems (digestive, cutaneous, vascular, etc.), in fuller detail and methodically.

In conclusion, although the diagnosis of small-pox is at times easy, there are occasions upon which it is most difficult, and no one sign is to be absolutely relied upon. Cases such as acne with granular kidney, chlorosis with backache, septic rashes from causes unascertained, syphilis in a rheumatic subject, a case of cerebral tumour, taking KI. or KBr. with vomiting—all these may be most misleading.

In cases where small-pox is present, however, it is often found that though the patient admits having had previous attacks of the same kind, yet the present is the first occasion on which he has for this cause abstained from work, laid up at home, or "had a doctor at home."

The whole article is well worthy of careful study.

Xylol has been recommended in the treatment of small-pox. Abbott[1] records a case treated successfully with it in India. The dose varies from 10 to 60 drops in milk, the larger quantities being given in three doses daily.

Nesfield,[2] influenced by the resemblance of small-pox to syphilis, and especially to the acute secondary form of the latter, tried treating the former condition by large doses of mercury with chalk. In all he treated eight cases—seven adults and a boy. To the former he gave 10 grains of mercury with chalk three times a day for six days, then twice a day for four days, and once a day for four days more—14 days in all. Although the number of cases treated does not justify any definite expression of opinion, he concludes :—

1. Ten grains of mercury with chalk three times a day by the mouth, continued for six days, produces no symptoms of poisoning in small-pox.

2. The drug appears to have a marked action in modifying and reducing the severity of the disease.

Snake Bite. Most of the papers on this subject are of a highly technical character and need not be considered here. Eight species of poisonous snake are now known in the Sudan, and these will be fully described and illustrated in a forthcoming work by Professor Werner, of Vienna. This authority has, however, very kindly furnished a short but useful account of these and other Sudan snakes, based partly on material collected by Dr. Wenyon (Third Report). Snake bite is not at all common in the Sudan. I have made many enquiries on this point, and have been invariably informed that cases are not often seen. *Naja nigrocollis*, the spitting cobra of the Sobat, is one of the most dreaded by the natives, and, thanks to the kind help of Dr. McLaughlin, of the American Mission of the Sobat, a sample of the venom, or rather of the ejected fluid, was obtained and sent to Professor Sir T. R. Fraser, of Edinburgh. Unfortunately, the quantity was so small that, though it was found to be active, Professor Fraser was unable to carry out any extended observations upon it. One hopes to secure a larger quantity later on, and applications for samples has been made in different quarters. It is acid and the antidote is an alkaline wash.

Rogers[3] has a paper on the treatment of snake bites. He divides poisonous snakes into two classes as regards their physiological actions. 1. Colubrine including certain sea snakes, the cobra and the krait of India : 2. Viperine including the African puff-adder, the Indian daboya, etc. (true vipers), and other rattlesnakes, etc. (pit vipers). The venom of the first class kills by paralysing the respiratory centre, that of the second by paralysing the vaso-motor centre.

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[1] Abbott, S. H. L. (May, 1906), "Xylol in Small-pox." *Indian Medical Gazette*, Vol. XLI.

[2] Nesfield, V. B. (April 25th, 1908), "On the Treatment of Small-pox by large Doses of Mercury and Chalk." *Lancet*, Vol. I.

[3] Rogers, L. (September 17th, 1904), "The Treatment of Snake Bites." *British Medical Journal*, Vol. II.

Snake Bite —*continued*

He deals with the action of antivenene, which he believes should be used intravenously, and then passes on to speak of the local treatment with permanganate of potash. He points out that it can only be used with advantage locally, being supplemented in the case of colubrine poisoning by antivenene used intravenously when it is available; in the case of viperine poisons, by adrenal extract repeatedly injected subcutaneously. The latter is valuable owing to the additional action of viperine venom on the blood, which it renders incoagulable, causing hæmorrhages from the bowel and into the tissues.

The local method is more fully described in another paper,[1] as is the special instrument and receptacle designed by Brunton and now so well known. A free opening should be made on the site of the bite and the crystals of permanganate rubbed into it after the limb has been tightly bandaged higher up. A few drops of saliva may be added to assist solution.

Much information will be found in various papers by Lamb.[2] The most recent standard work on the subject is Calmette's "Les Venins," etc., but exception is taken to his views as regards specificity in an interesting critique,[3] which also refers to an article in Allbutt's "System of Medicine," Vol. II. Part II., as upholding the conclusion that both *in vivo* and *in vitro* the venoms are highly but not strictly specific.

A note may be made of the Brazilian method[4*] of von Bassewitz, which consists in removing the gall bladder of the venomous snake, triturating it in physiological salt solution, filtering and injecting the filtrate under the skin of the back or flank. The results are said to be excellent, but the method should only be used in cases where freshly-prepared serum is not available. Abscesses are apt to occur at the site of injection.

Spider Bite. Information is required regarding this condition in the Sudan. Fink[5] has an interesting note upon it with reference to India. He says:—

> The effect of a certain spider lick or bite is to be seen frequently in children in Bengal, when the face is generally the part affected, owing to this part of the body coming frequently into contact with spider webs. The result is often an eruption on the lips or chin, resembling *Herpes*, which, if untreated, goes on to resemble *Impetigo contagiosa*, if you compare these eruptions with it.
>
> *Treatment.* The most effectual treatment of a spider's lick or bite is as follows:—
>
> Take a basin of cold water and let the patient hold his or her head over it. Now get a few lumps of mustard oil cake (which is to be had in almost every oilman's shop, since the cake is obtained after expressed mustard oil is manufactured, and is used largely to fatten cattle), burn these in a charcoal fire till of a black colour. Drop the burning lumps into the basin of water, and allow the smoke and fumes which rise to come into contact with the part of the patient's skin which is affected, twice a day, for two or three days. The result is a perfect cure.

Spirochætes and Spirochætosis (including Relapsing Fever). These subjects have recently attained such great dimensions that it will not be possible to do anything like justice to them. A comparatively brief review must suffice.

Under the special paper on "Spirochætosis of Sudanese Fowls" (Third Report), various points omitted here will be found discussed.

Leishman[6] gives Lühe's classification of spirochætæ in three groups, as follow:—

A. True Spirochætæ

1. *Spirochæta plicatilis.* The type species. A water organism which may reach a length of 200 μ, and in which Schaudinn has demonstrated the presence of an undulating membrane and the absence of flagella.

2. *Spirochæta balbianii.* Found in the intestine of the oyster, and recently re-investigated by Perrin, who figures and describes remarkable details of the structure, and definitely classes it as a trypanosome.

3. *Spirochæta dentium.* A very minute spirochæta found in the mouth and dental tartar of man.

4. *Spirochæta buccalis.* A larger organism, found in the same situation as *Spirochæta dentium.*

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[1] Brunton, Sir L., Fayrer, Sir J., and Rogers, L. (September, 1904), "A Method of Preventing Death from Snake Bite, capable of Common and Easy Application." *Indian Medical Gazette*, Vol. XXXIX.

[2] Lamb, G., "Scientific Memoirs by Officers of the Medical and Sanitary Departments of the Government of India." Nos. 1, 3, 10 and 17.

[3] *Indian Medical Gazette*, September, 1907, Vol. XLII.

[4] Annotation in *British Medical Journal*, 24th December, 1904, Vol. II.

[5] Fink, G. H. (December 1st, 1906), "Peculiar Eruptions of the Skin in India, due to Vegetable and Insect Life and their Treatment." *Journal of Tropical Medicine and Hygiene*, Vol. XIX.

[6] Leishman, W. B. (September, 1906), "Pathogenic Spirochætæ." *Journal of Preventive Medicine*, Vol. XIV.

* Article not consulted in the original.

pirochætes nd Spiro- ætosis— *continued*

5. *Spirochæta vincenti.* Found by Vincent in a pseudo-diphtheritic condition of the throat, and also in hospital gangrene.
6. *Spirochæta vaccinæ.* Found in vaccine lymph.
7. *Spirochæta refringens.* In syphilitic and other lesions of skin and mucous membranes.
8. *Spirochæta pseudo-pallida.* Often found in association with *Spirochæta pallida.*
9. *Spirochæta of dysentery.* Described by Le Dantec.

B. Blood Spirochætæ

1. *Spirochæta anserina.* Found in geese, by Sakharoff.
2. *Spirochæta gallinarum.* From fowls in Brazil, by Marchoux and Salimbeni.
3. *Spirochæta obermeyeri (vel recurrentis).* The cause of relapsing fever.
4. *Spirochæta of tick fever.*
5. *Spirochæta theileri.* From Transvaal cattle.

In addition to the above, spirochætæ have been found in the horse, sheep, bat, Norway rat, bandicoot, and also in the cockroach, mosquito, and tsetse fly, which may, probably, on further knowledge, be included in this group of blood spirochætæ.

C. Treponema

1. *Treponema pallidum.—Syphilis.* Schaudinn and Hoffmann.
2. *Treponema pertenuis.—Yaws.* Castellani.

He then gives notes on certain of the pathogenic forms citing the work of Karlinski as showing that probably the common bed-bug is the carrier of *S. obermeyeri.* He mentions the work of Novy and Knapp, who were successful in keeping the spirochætæ alive through many generations in both white rats and mice by intra-peritoneal inoculation. He has not been able to confirm the presence either of the undulating membrane of Schaudinn or the flagella of Novy and Knapp, and while he considers it quite possible that European relapsing fever may differ from that found in Bombay he is not prepared to uphold the morphological distinctions put forward by these latter authors.

As regards African tick fever, he says :—

This disease appears to be widely distributed in Africa, occurring in Uganda, the Congo Free State, and in German East Africa. The spirochæta was found by Ross and Milne, and, independently, by Dutton and Todd, who further proved that it was transmitted by a tick, the *Ornithodoros moubata.* The young of infected ticks, reared in captivity, were also shown to be infective, and Koch has recently been able to trace the spirochætæ up to a certain point in the development of the embryo. The question as to the identity of this tick fever with the relapsing fever of Europe is the subject of much debate, and, although Koch holds them to be identical, there are many points of difference as regards symptoms, fatality, animal reactions, etc., which make it hard to accept this view. Again, while Koch regards the spirochætæ as morphologically indistinguishable, Novy and Knapp describe many points of difference, and suggest for the tick fever spirochæta the name of *Spirochæta duttoni* in memory of Dutton, who lost his life in the investigation of the disease.

In this connection the monkey spirochæta, is of interest, as it was found by Gray and Tulloch, at Uganda, in the blood of a monkey naturally infected. It corresponds to the organism of tick fever in morphological appearance, and it is not improbable that it is identical with it.

A somewhat similar paper is that by Blanchard.[1] Some spirochætæ described by Leishman he does not mention, but, on the other hand, he notes *S. eberthi,* found in the intestines of various birds. *S. ovina,* found in the blood of an Abyssinian sheep (*see page* 87), and spirochætæ which occur in the stomach of dogs, were found by Bizzozero, and appear to possess flagella. A different form of spirochæte was found by me[2] in the stomach and intestines of dogs and other animals dying of experimental trypanosomiasis in the Sudan.

A more recent article is that by Mühlens,[3] who mentions the spirochæte of carcinoma and that found in gangrene of the lung.

Fantham,[4] in a general paper, speaks of *S. culicis* (Jaffé) from the alimentary tract of a mosquito, *C. pipiens* and *S. bufonis* (Dobell) found in the rectum of a toad.

Bousfield[5] has described in the Sudan cases of membranous ulceration of the throat associated with the presence of spirochætes. He regards the condition as being allied to,

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[1] Blanchard, R. (1906), "Spirilla, Spirochætes and other Spirally-formed Organisms." *Revue Vétérinaire.* Quoted in *Journal of Tropical Veterinary Science,* July, 1906, Vol. I.

[2] Balfour, A. (1906), "Second Report of Wellcome Research Laboratories."

[3] Mühlens, P. (September 28th, 1907), "Vergleichende Spirochatenstudien." *Zeit. für Hygiene u. Infekt. Krankheit.,* Bd., LVII., No. 3.

[4] Fantham, H. B. (July, 1908). *Science Progress,* Vol. III.

[5] Bousfield, L. (September 14th, 1907), "Observations from the Sudan." *Lancet,* Vol. II.

Spirochætes and Spirochætosis—*continued*

if not identical with, that described by Vincent, but notes certain differences between his three tonsillar cases and those described by that author. These were:—

1. Fœter was evident in Vincent's cases, while this was absent in all enumerated in Bousfield's article. 2. Vincent's cases were practically all unilateral, while of Bousfield's, two out of three were bilateral. This point is of importance, for H. W. Bruce states that mild cases are invariably unilateral. 3. Vincent mentions that bacilli fusiformes were sometimes unassociated with spirochætæ and often outnumbered them. In these cases the two were always associated with one another, and the spirochætæ usually enormously outnumbered the bacilli. 4. Pyogenic cocci were almost invariably present in Vincent's cases, while in several cases of this series the infection was practically pure with spirochætæ and bacilli fusiformes. 5. Vincent mentions extension to the soft palate and uvula, and in none of Bousfield's cases was this present, perhaps owing to the cases being seen early in their course. 6. Vincent makes no remark about the urine, but Bruce mentions albuminuria in one out of ten of his cases. Albumin was present in two out of three cases of tonsillitis mentioned in Bousfield's article.

Some of the latter's cases showed ulcers in the buccal cavity. Bousfield has also found spirochætes in destructive ulcer of the penis along with *B. fusiformis* and cocci.

Schellach[1]* has made important researches on the spirilla of European, African and American relapsing fever.

In order to obtain exactly comparative results, very careful precautions were taken by the author, the spirilla being observed in the living state always in the first stage of injection in rats, mice and monkeys, while the stains and fixations employed were always applied under uniform conditions.

The flexibility and activity of movement of the Russian species are greater than in the American, but less than in the African species. Very exact details are given of the length and regularity of the spirals, and the characters of the motion, corkscrew-wise, lateral bending, bending through a circular arc, and the special movements of the ends of the spirals.

The thickness of these species is especially difficult to measure, but the author gives the diameter in microns as 0·45, 0·31 and 0·39 for the African, American and Russian forms, respectively, in specimens stained by Giemsa's method. The African form is about 24 μ at the most, with 8 to 12 undulations; the American, 17 to 20 μ with 6 to 8 undulations; and the Russian, 19 to 29 μ, with 8 to 10 undulations.

In spirochætes, whose vitality has been diminished by washing and centrifuging, or by prolonged keeping in serum, the spirals are more rigid and regular, and seem dependent on the structure of the organism.

The author believes that in all three forms transverse division is the only method; Y-shaped forms are exceptional, and can only be regarded as an appearance due to commencing division. Forms may also be met with still, joined by a thin filament of protoplasm, with, for example, in *S. duttoni*, only four to six, or half the adult number of undulations, whereas the full number of turns would be preserved if the method of division were longitudinal.

One of the ends of the spirochætes terminates in a filiform prolongation some five μ long, which grows as the rest of the organism does, while the other, though pointed, ends abruptly. The existence of a lateral membrane could not be verified in either species; like Fränkel, he was able to demonstrate lateral cilia by Zettnow's method, but considers them to be the artificial result of the numerous washings, centrifugings, etc., involved in the method.

Deep staining by Giemsa's method brings out certain coloured granules, but no difference between the species could be demonstrated in this respect.

Lastly, some six pages are devoted to the distribution of the parasites in the organs and tissues of infected rats, and the conclusion is reached that, unlike *S. pallida*, which is a tissue parasite, the spirochætes of relapsing fever are true hæmatozoa. These bring about necrosis of the liver and spleen, but in the intervals between the attacks are not quartered specially in the spleen, but equally divided amongst the organs generally.

Like Manteufel, he is sceptical as to the destruction of the parasites taking place by phagocytes, but states that extra-cellular destruction is effected in the liver and spleen. Lebert proposed, in 1874, the name of *Protomycetum recurrentis* for the Russian species, while Colin, in 1875, proposed that of *S. obermeieri*.

According to the rules of nomenclature, therefore, the name should stand as *S. recurrentis* (Lebert). The African species should stand as *S. duttoni* (Novy), while for the American the author proposes *S. novyi*, which, if accepted, would thus stand *S. novyi* (Schellach).

Manteufel,[2]* working with the spirilla of relapsing fever, found apparently that the spirochætes can penetrate the shaven but unbroken skin of a rat and produce infection. He conducted agglutination experiments and found that, contrary to what is seen in bactericidal infections, a serum which is extremely active in agglomerating living and active spirilla is quite inert in the case of the same spirilla which have been killed by heating to 45° C.

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[1] Schellach, C. (1907), "Morphological Researches on the Spirilla of European, African and American Relapsing Fever." Arb. a. d. *Kaiserl Gesundheitsamte*, Bd. XXVII., Heft. 2. Quoted in *Journal of Tropical Medicine and Hygiene*, March 16th, 1908, Vol. XI.

[2] *Loc. cit.*

* Article not consulted in the original.

Spirochætes and Spirochætosis— *continued*

Leishman,[1] reviewing papers read at the Berlin International Congress of Hygiene, gives Doflein's views. The latter regards present staining methods as inadequate for pathogenic spirochætes, and has not much belief in the reputed presence of a blepharoplast. He thinks spirochætes vary as to their mode of multiplication, and he is inclined to class them in a special group, midway between the animal and the vegetable kingdoms, for which he suggests the name "Proflagellata." Levaditi pointed out that all the pathogenic spirochætes, except those of syphilis, yaws and a dermatosis of the pig, are the cause of true septicæmias. He thought their protozoal nature had not been proved, but was inclined to attach some importance to the reported discovery of peritrichic flagella in *S. gallinarum* and *S. duttoni*.

As regards the mechanism of relapses, Levaditi held to the opinion long ago expressed by him that the crisis is due to phagocytosis of the spirochætæ and their intra-cellular digestion, and not through the action of specific anti-bodies, which only make their appearance in the blood after the crisis is over. He pointed out that the spirochætæ did not, however, disappear entirely from the blood during the interval (*see*, however, special paper on "Spirochætosis of Fowls," Third Report), but that a few could always be found on careful search. He believed that such spirochætæ as had escaped destruction during the crisis had become immune to the action of the anti-bodies, and in this way were able to multiply and produce a second attack.

He had found that such immune spirilla were able to transmit their powers of resistance to their progeny, because relapse-spirochætæ were found to preserve their resistance after several passages through susceptible animals.

Vaccination can easily be produced in animals by the injection of killed spirochætæ, or of a non-lethal dose of living organisms. Preventive serum-therapy is also possible, because the serum of animals which had recovered was found to be strongly bactericidal and agglutinative.

The reader may also consult a short French paper by Borrel,[2] which discusses the relationship of the spirilla, spirochætæ and trypanosomes, and gives illustrations of the terminal flagella in *S. gallinarum* and the so-called undulating membrane of *S. balbiani*.

Passing from these more or less general papers, we may first consider papers on African Spirochætosis, then take up the Indian, European and American varieties, in each instance citing observations as regards the vector, the parasite, the clinical symptoms, preventive measures and treatment.

Koch's[3] observations are thus summarised in the *Medical Annual*, 1907. He notes with regard to the habits of *Ornithodoros moubata*, which transmits the spirochæte *(S. duttoni)* of African tick fever, that it is exclusively a human tick. It is nocturnal in its habits, and after sucking blood, quickly hides again in the earth of the native huts or rest-houses. It likes dry soil, and in fact, if the earth is moist (as is the case when goats are brought inside the hut at night) no ticks are to be found. The African tick fever differs clinically from the European lapsing fever *(S. obermeieri)* in the following points. In the European form the first attack lasts six to seven days; then follows an apyrexia of five to six days, then a second somewhat shorter attack and a longer apyrexia of five to six days, then a second somewhat shorter attack and a longer apyrexia, and so on. In the African form, the maximum duration of the attack is three days and the apyrexia six to ten days. Again, in the African form the number of parasites may be extremely scanty, and thus difficult to find. It is advisable always to make a smear as large as a sixpence, dry thoroughly, dehæmoglobinise with water (without fixing), and then stain with gentian violet, five to ten minutes.

Koch has found spirochætes in about a quarter of the eggs up to the twentieth day of development of ticks which had sucked spirochæte blood. They can then be seen no longer, but must exist, as young ticks carry the infection. It is probable, indeed, that ticks in the young stage are by far the most infective (though adults also may convey the disease). Koch found infected ticks to occur in practically every hut examined, in a variable proportion, *e.g.* 7 to 50 per cent. In the huts, however, there are frequently no natives suffering from spirochæte fever. The question, therefore, arises, how do these ticks get their infection? Various hypotheses are possible, of which only one may be mentioned here, viz., that rats contain spirochætes, and that they may act somewhat as rats do in

[1] Leishman, W. B. (April, 1908). *Journal of the Royal Army Medical Corps*, Vol. X.

[2] Borrel, A. (March 2nd, 1908), "Spirilles, Spirochètes, Trypanosomes." *Bull. Soc. Path. Exot.*, Vol. I.

[3] Koch, R. (February, 1906). *Berl. Klin. Woch.*

Spirochætes and Spirochætosis—*continued*

regard to plague. The natives in tick districts have a considerable immunity, probably through attacks in youth, as monkeys that have had a severe attack are quite immune against a new infection. Infection can generally be easily avoided by not sleeping in native huts or rest-houses, the favourite haunts of the ticks.

A very good account of the disease in Uganda is given by Moffat.[1] He believes it to be distinct from the tick disease of the Zambesi, and states that he has never seen the incubation period less than seven days. One attack confers a certain degree of immunity, and after a second attack this immunity probably becomes complete, at least for a time.

The number of relapses seems to vary from one to five. Moffat points out that in a susceptible subject the initial attack is followed by from three to five relapses, and that if only two relapses occur he suspects a partial degree of immunity.

In the earlier attacks parasites are generally more numerous than in the late ones, in which they may be excessively rare, but this does not always hold good. The symptoms are described and special attention drawn to a curious condition affecting the eyes. It resembles an iritis, but is possibly a condition resulting from thrombosis in the vessels of the ciliary body and iris, leading possibly to hæmorrhages into the vitreous. At times it does develop into an acute iritis with resulting adhesions of the usual kind. The portion of the paper dealing with diagnosis is given in full, as there is every probability that this disease exists in the Southern Sudan, although hitherto unrecorded. It is true we have not yet found *O. moubata*, but the closely allied *O. savignyi* occurs.

The spirochætæ can easily be found in fresh blood if present in large numbers, but a thin, evenly-spread film is quite unsuitable, and in such they may be overlooked even when numerous. A film of moderate thickness is preferable, and in such the agglomerated masses of corpuscles will sometimes be seen vibrating from the movements of the contained parasites if the latter are sufficiently plentiful, and by a careful focussing they may be seen semi-detached wriggling in the surrounding serum. Being very refractile they are best seen with little light and appear as rapidly moving threads; sometimes they may be made out quite distinctly moving over and against the dark background of a thick layer of corpuscles. There is no doubt, however, that they are much more easily found in stained preparations. I have tried the two methods side by side in the same case, and after a vain search through the fresh film I found them at once in the stained one. The quickest way is to take a fresh film and a dry one at the same time. A few minutes are devoted to the former and, if the parasites are numerous, they will at once be seen and the trouble of staining is avoided. If the search prove negative, staining can be proceeded with. I have for the most part used Leishman's stain, which is simple and effective, and the parasites can easily be seen under a sixth. At times they are very sparse, necessitating a prolonged search through several slides. In the absence of means for making a blood examination, it is not possible to make an absolutely definite diagnosis. A history of previous tick bites, or of a possible exposure to such, will afford strong suspicion as to the nature of the fever, but in places where the disease is endemic there are many other biting things, and people are so accustomed to their attacks that very often there will be no distinct remembrance of such. The disease with which it is most likely to be confused is malaria. In its onset it differs from it in the absence of any rigor, though it must be remembered that in the malignant tertian the rigor is represented often by a slight and evanescent feeling of chilliness, but otherwise the initial symptoms are very much alike, though, as a rule, they are more aggravated in spirillum fever, especially the headache and vomiting. In the greater number of cases of malaria there is no particular enlargement or tenderness about the liver, whereas in spirillum fever they are almost constant features. The splenic enlargement is common to both. The appearance of the tongue, with its thick, creamy white deposit of fur, is quite unlike the dirty, dry tongue of malaria. As regards the temperature, there is usually in malaria a distinct tendency to intermission or, at any rate, a marked remission, such being generally preceded by profuse sweating. Spirillum fever attacks are sometimes so short that a sudden termination cannot be attributed to the influence of any remedy administered, and therefore the action of quinine on the temperature cannot be relied upon as a means of diagnosis, except to exclude malaria when the drug fails. The quick breathing and pains in the chest seen in spirillum fever do not occur in malaria. In the pneumonic form a diagnosis can only be made by watching the course of the disease, but it may be noted that the physical signs in the chest not only disappear, but develop much more rapidly than they do in an ordinary pneumonia.

The eye symptoms point very strongly towards spirillum fever. I formerly believed that malarial iritis was a common complication of that disease, but since I began to depend for diagnosis on blood examination I have not seen a case of iritis following malaria. Probably in my earlier cases the antecedent fever was spirillum.

The prognosis is not unfavourable as regards life, but the disease causes much suffering and leaves the patient greatly debilitated. Pneumonia is a dangerous complication. There is no specific treatment, but arsenic possibly diminishes the number of relapses. Symptomatic treatment is indicated, and the exhibition of atropine on the first sign of eye trouble.

In this connection one may note that benzidine has proved useful in experimental tick fever, and pyramidon has been found to reduce the temperature, though it leaves the spirochætæ unaffected. Judging by analogy, atoxyl is likely to prove of value, but Breinl and Kinghorn found it useless in one case.

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[1] Moffat, R. H. (January 26th, 1907), "Spirillum Fever in Uganda." *Lancet*, Vol. I.

Spirochætes and Spirochætosis— *continued*

Ross[1] also describes Uganda "Tick Fever," giving several temperature charts. He differs in one or two points from Moffat, finding that there may be as many as six relapses in the European, and that in the native no relapse may occur. He also notes that the parasites are usually exceedingly sparse in the blood and that it may take many hours' search to find one organism. Where blood examination fails the diagnosis can be made with ease and certainty by injecting a drop or two of finger-blood into a monkey. When the animal sickens the spirochætæ can be found with the greatest ease in the blood. This method, however, takes a few days. It is worth noting that there is a relative increase of polymorphonuclear leucocytes.

As regards prevention, this is easy for Europeans but difficult in the case of natives. The *Ornithodoros* lives by day in the thatch or in cracks of mud-floors and walls of old native huts. At night it comes out in search of food, retiring again when it has fed. Old camping grounds and old huts should be avoided, infected huts should be burned. Europeans should sleep under mosquito nets, well tucked under the bed-clothes. The liability of natives to relapses is possibly due to repeated re-infection.

An account of the disease as it occurs in Angola is given by Wellman.[2] An important experimental study on *Spirochæta duttoni* was carried out by Breinl and Kinghorn.[3] As regards technique, they state :—

The routine method of examining the blood for spirochætes was the ordinary thick-film one. Two or three drops of blood were placed on a perfectly clean slide and then spread out over a surface 2 c.c. by 3 c.c. After drying in the air, the films were fixed in the flame in the same way as a bacteriological specimen, and the hæmoglobin was removed by washing the films in distilled water. After being so treated, they became quite colourless and were then stained with Romanowsky's stain for half an hour. The stain was made in accordance with the directions given by Stephens and Christophers.

A.	Medicinal methylene blue,	1 part
	Sodium carbonate,	0·5 „
	Distilled water,	100· „
B.	Eosin,	1:1000· „

Before using, dilute each solution with 19 parts of distilled water, and then mix in equal parts for staining. This method gave us better results than any other modification of Romanowsky.

In specimens prepared by this method the spirochætes are well defined and are of a deep purple colour. The leucocytes are well stained, while the red cells appear as mere shadows. The examination is much facilitated by reason of this.

For more detailed study, very thin films were made on slides heated to 37° C., in order to dry the blood-film more rapidly; these were fixed in absolute alcohol and stained with the above modification of Romanowsky and by Giemsa's and Laveran's method. In our hands Marino's method did not yield satisfactory results.

When the presence of precipitates interfered with examination, it was found advisable to place the preparation in oil of cloves for a short time, and then in xylol after the excess of oil had been blotted off.

Carbol-fuchsine stains the spirochætes very readily and intensely, but is not as valuable a stain as that of Romanowsky. Heidenhain's iron-hæmatoxylin was also used, but without any advantage, as it stains the spirochætes uniformly black.

In order to study the structure of the parasite the wet film method was used. Perfectly clean slides were covered with an exceedingly thin layer of Mayer's albumen. A drop of blood was spread out as quickly as possible over the layer of albumen, and while still wet the slide was dropped into Flemming's fixing solution and left for ten minutes. In this the albumen was quickly coagulated and firmly fixed the blood to the slide. From the fixing solution the slide was passed through the different alcohols and stained.

The routine examination was made with a Zeiss $\frac{1}{12}$th oil immersion and No. 4 ocular.

They found by experiment that the blood of patients suffering from relapsing fever is infective for susceptible animals during the period of apyrexia. They compared African tick fever and European relapsing fever, and agreed with Koch as to the shortness of the attacks in tick fever, and as to the small number of parasites in the blood as compared with European relapsing fever. The attacks and relapses are of longer duration in the latter.

In the study of the animal reactions they found that they could infect nearly all the usual laboratory animals with *S. duttoni*. Cats were refractory to infection. The most susceptible animals were white rats and then monkeys.

As regards immunity, they note that there is a relatively active immunity against re-infection, as animals re-inoculated at various intervals after recovery up to seven-and-a-half months did not become infected at all or only had a very slight attack.

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[1] Ross, P. (March 1st, 1906), "Tick Fever." *Journal of Tropical Medicine and Hygiene*, Vol. IX.

[2] Wellman, F. C. (April 1st, 1905), "Case of Relapsing Fever, with Remarks on its Occurrence in the Tropics and its relation to Tick Fever." *Journal of Tropical Medicine and Hygiene*, Vol. VIII.

[3] Breinl, A., and Kinghorn, A. (September, 1906), "An Experimental Study of the Parasite of the African Tick Fever (*Spirochæta duttoni*)." *Mem. Liv. Sch. Trop. Med.*, No. XXI.

Spirochætes and Spirochætosis—*continued*

As regards treatment, their experiments showed that immune serum, whether derived from horses, monkeys or rats, has no appreciable value either in preventing the occurrence of the attacks in susceptible animals or in curing the disease once contracted. The incubation period may be prolonged to a greater or less extent, but the inoculation of infective blood is always followed by infection.

In one case hyper-immune serum, *i.e.* serum derived from animals after a varying number of inoculations with spirochætal blood, used as a preventive, prolonged the incubation period very markedly and moderated the severity of the attack. Similar horse serum used as a curative agent proved itself of no pronounced value in the treatment of the disease in monkeys. A slight inborn immunity of short duration was noticed.

Other points elucidated were that the spirochæte of African tick fever is of a species differing from *S. obermeieri*, since each confers a relatively active immunity against itself but not against the other, that *S. duttoni* passes through the placenta from the circulation of the mother to that of the fœtus, that the course of the disease in spleenless animals does not differ in any way from that noted in normal animals, and that spirochætes when disappearing from the blood do not rest solely in the spleen.

Experiments of tick feeding and splenectomy during the incubation period showed that spirochætes are present in the peripheral circulation in an infective stage on the first day after ticks are fed on a susceptible animal, and that :—

1. In splenectomised animals, the spirochætes disappear from the peripheral circulation after the attack as promptly as in normal animals and relapses occur in the ordinary way.

2. When the spleen is removed shortly after the spirochætes have disappeared from the peripheral circulation after the first attack, the relapses occur as in the controls.

3. During the incubation period, after ticks have been fed on a susceptible animal, the spirochætes do not develop in the spleen as the site of election.

4. Active immunity against reinfection is not influenced by the spleen.

Dealing with stained specimens, they note that the terminal flagellum of some observers is the periplast of the parasite drawn out to a pointed extremity at one end of the spirochæte, that the chromatic core does not stain evenly, and that peculiar forms are seen most often in the "decline" blood. The core may be broken up into from six to eight small portions, which stain deeply by Giemsa's method. In films made from the liver and spleen they found a curious form in the shape of a spirochæte coiled up into a small compass surrounded by a well-stained membrane, the whole structure being about three-quarters the size of a red blood cell. They think this may be an encysted stage.

Together with Todd,[1] these authors also found that *Cimex lectularius*, the bed-bug, is probably unable to transmit *S. duttoni* or *S. obermeieri*, and therefore it cannot be an important factor in the causation of epidemics of relapsing fever.

Breinl[2] has further pursued the study of the morphology and life-history of *S. duttoni*. He employed dry films and the Giemsa stain. Wet films were found to present no advantages. He failed to demonstrate peritrichal flagella, and notes the frequent presence of swellings during the stage of "decline," as also the presence of a small unstained transverse band at about one-third of the length of the parasite. No true undulating membrane could be demonstrated. The division of *S. duttoni* was, as a rule, found to be transverse, though occasionally longitudinal division was observed, as was also an appearance suggesting conjugation.

Allusion is made to Prowazek's discovery of intra-cellular stages of *S. gallinarum* in the red blood cells (*see* also special article on "Spirochætosis of Fowls," Third Report), and it is noted that the same phenomenon in rare instances was observed just before the crisis in *S. duttoni*. The formation of coiled and skein-like forms at this time in the spleen and bone marrow, rarely in the liver, is described as is the phagocytosis which they undergo in the spleen, or, if the spleen be removed, in the liver. Some, however, escape destruction and become surrounded by a thin cyst wall, the interior of the cyst being filled with a faintly blue-stained plasma. The shape of the parasite becomes indistinct until only a few small red granules persist. These may be the forms which can pass through a Berkefeld filter and give rise to a fresh infection. The life-history of the spirochæte is thus summarised :—"Just before the crisis the spirochætes disintegrate, certain of them coiling up into skeins, the

[1] *Loc. cit.*

[2] Breinl, A. (November 9th, 1907), "On the Morphology and Life-History of Spirochæta Duttoni." *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. I., No. 3.

Spirochætes and Spirochætosis— *continued*

majority of which are phagocytosed by the spleen. Some of them become encysted and break up into very small bodies out of which the new generation of spirochætes is evolved." A coloured plate illustrates the various appearances mentioned.

Loghem,[1] following up this work, and using Levaditi's method for the organs of infected rats, found at the height of infection atypical forms in the liver and spleen. These can be recognised only by their staining reactions, by the conditions under which they are found, and by the presence of intermediate stages. They occur as small, circular, or oval, tightly-coiled spirals, about half the size of a red blood cell. These are the encysted forms of Breinl and Kinghorn, the "agony forms" of Levaditi, preceding further stages of degeneration and disintegration, the "resting stages" of Prowazek, who takes quite a different view of them. They were most commonly seen in the liver, and usually within the phagocytes, not in the lumen of the capillaries. Loghem thinks Levaditi is correct in his supposition that these forms are due to the influence of phagocytosis. A French paper by Levaditi and Manouélian[2] deals with much the same questions, and may be consulted with advantage.

Carter[3] investigated the presence of *S. duttoni* in the ova of *Ornithodoros moubata*, and concludes that the ticks infected by spirochætes lay infected eggs, that multiplication of the spirochætes probably takes place in the eggs, and that morphological changes in the spirochætes also occur in the eggs. Illustrations of the curious banded and swollen forms met with in the eggs of the tick are given. Möller's work (Berlin, 1907), with monkeys, has shown that a tick may remain infective for one and-a-half years and that the infection may be conveyed to the third generation of ticks.

Rogers[4] describes the Indian form and mentions that Sandwith has observed the disease in Egypt. The latter author,[5] indeed, describes Egyptian relapsing fever at considerable length, noting that it is more common in the Delta than in Upper Egypt or the Sudan, and mentioning its tendency to occur along with typhus. The symptoms of the Indian fever are given very fully by Rogers, and amongst special symptoms one notes slight enlargement of the liver and of the spleen, jaundice, and a rash which occurs in about 10 per cent. of the cases. The rash appears on the front or sides of the chest or abdomen, or on the arms and more rarely on the legs, and is in the form of clusters of minute red blotches or stains.

Atypical forms of the fever occur, simulating malaria and typhus fever. The latter, constituting usually fatal forms, have been described by Carter as "Icteric Fever," owing to the presence of intense jaundice. Such cases might be mistaken for yellow fever and Weil's disease. Lowenthal's reaction is mentioned as an aid to diagnosis during the intervals when there are no spirilla in the blood. A drop of blood from the suspected case is mixed with another drop containing spirilla from a case in the febrile stage, sealed under a cover glass and incubated at 37° C. for at least half an hour. Clumping and loss of motility indicate relapsing fever, provided a control is negative after not more than two and a half hours.

The work of Mackie on bed-bugs as carriers of the disease is quoted as follows :—

> Quite recently the question of the mode of infection of relapsing fever in Bombay has been investigated by F. Percival Mackie, I.M.S. On examining bed-bugs at various intervals after being fed on monkeys infected with the disease, the spirilla was found up to the fourth and seventh days respectively in two series of experiments in the upper part of the alimentary tract only, together with fresh blood. Out of fifty-three bugs from the relapsing fever ward, spirilla were only found in the stomach of one. Of six monkeys, into whose cages bed-bugs were frequently placed, only one contracted the disease. Dr. Mackie also informs me that the disease can be transmitted by the punctures of a grooved needle carrying a trace of fresh blood, so that it appears to be not unlikely that mosquitoes might thus convey it from one patient to another, which would account for the infection in wards not being most commonly between neighbouring beds, as Vandkye Carter pointed out. For this reason mosquitoes may be a more likely carrying agent than bed-bugs, and are worthy of close attention. The disease was also transmitted by oral feeding with slightly larger quantities of blood, the incubation stage being prolonged to four or five days. Blood drawn during an apyretic interval, with no spirilla in the blood, also infected monkeys, the fever developing late at the same time that a relapse occurred in the man from whom it had been taken."

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[1] Loghem, J. G. van (February 29th, 1908), "Some Notes on the Morphology of *S. duttoni* in the Organs of Rats." *Annals of Tropical Medicine and Parasitology.* Series T.M., Vol. I., No. 4.

[2] Levaditi, C., and Manouélian, Y. (April 25th, 1907), "Recherches sur l'infection provoquée par le Spirille de la Tick-fever." *Ann. de l'Institut Pasteur*, t. XXI., No. 4.

[3] Carter, R. M. (February 1st, 1907), "The presence of *S. duttoni* in the Ova of Ornithodorus moubata." *Annals of Tropical Medicine and Parasitology.* Series T.M., Vol. I., No. 1.

[4] Rogers, L. (1908), "Fevers in the Tropics."

[5] Sandwith, F. M. (1905), "The Medical Diseases of Egypt." Part I.

Spirochætes and Spirochætosis—*continued*

Mackie[1] suggests the name *Spirillum carteri* for the Indian parasite.

Later work[2] by the same author is of great interest and importance, for it goes to prove that the body-louse, *Pediculus corporis*, is probably a transmitter of the disease. He investigated an outbreak at a mission settlement in India and summarises his investigations and conclusions as follow :—

1. An epidemic of relapsing fever broke out in a mixed settlement of boys and girls living under similar conditions.

2. A very high percentage of the boys fell victims to the disease in the course of a few weeks.

3. A much smaller percentage of girls fell ill and at infrequent intervals extending over three months.

4. The most notable factor in which the boys differed from the girls was that they were infested with body-lice, from which parasite the girls were almost free.

5. A well-marked percentage of the lice taken from the infected ward contained living and multiplying spirilla.

6. The stomach of the louse was the chief seat of multiplication, and this was carried on in the face of active digestion and after the disappearance of all other cellular elements. Other organs became secondarily infected. The secretion expressed from the mouth of infected lice contained numbers of living spirilla, and they also existed in greater or less numbers in the upper alimentary tract. The ovary was frequently infected, but spirilla were not found in deposited ova.

7. With the increase of the epidemic amongst the girls, body-lice became more in evidence.

8. With the subsidence of the epidemic amongst the boys, the percentage of infected lice fell.

9. An attempt to infect a monkey by means of lice failed.

I think that the above facts are sufficient to throw grave suspicion on the body-louse as a transmitter of relapsing fever. Many epidemiological facts point to this mode of transmission as being a likely one, and the life-history and habits of body-lice as outlined above show how well these parasites fulfil the necessary conditions for spreading the disease.

Thus relapsing fever has always been associated with poverty-stricken, overcrowded, and half-starved communities, and it is under just such conditions that lousiness is at its worst. Again, in mixed communities, as in Bombay, the disease attacks the poor, dirty and low-caste living in squalid tenements, to the exclusion of those of cleanly habits and better conditions of life.

Relapsing fever is a "personal" and not a "place" disease, and among stricken communities the infection spreads from person to person very rapidly after only a few days' exposure, and mere contiguity without contact is not sufficient to carry on the infection.

It is probable that the infected secretion of the louse's mouth is the medium responsible for transmission during the height of an epidemic, but whether the ovarian infection plays any part in the linking together of epidemics or in the carrying over of the disease during the off season it is at present impossible to say, but the failure to find spirilla in nits is rather against this theory.

The same epidemic is described by Landon,[3] who also mentions the presence of lice, and states that in native circles relapsing fever is called "Lice Plague." He mentions the value of adrenalin chloride for heart-failure occurring in the course of the fever. It can be given in 1 : 1000 solution, as sold, in doses of 5 to 30 minims. In less urgent cases, 10 minims of 1 : 5000 solution every four hours were found to have a remarkable effect.

Sergent and Foley[4] have made observations, clinical and experimental, tending to implicate the clothes-louse, *Pediculus vestimenti*, in South Oran in Algeria. Their work, so far, is only of a preliminary character and need not be detailed.

Desai[5] deals with the clinical features of the Indian disease, which, he says, is frequently mistaken for plague in Bombay. He mentions pains in the calves as a prominent symptom constantly present, and speaks of the "air hunger" respirations which are relieved by quinine.

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[1] Mackie, F. P. (September 21st, 1907), "A Preliminary Note on Bombay Spirillum Fever." *Lancet*, Vol. II.

[2] Mackie, F. P. (December 14th, 1907), "The Part played by Pediculus Corporis in the Transmission of Relapsing Fever." *British Medical Journal*, Vol. II.

[3] Landon, E. (December, 1907), "Some Clinical Observations on Relapsing Fever." *Indian Medical Gazette*, Vol. XLII.

[4] Sergent, E., and Foley, F. H. (March 2nd, 1908), "Fièvre récurrente du End-Orunais et Pediculus vestimentis," note Préliminaire. *Bull. Soc. Path. Exot.*, Vol. I., No. 3.

[5] Desai, V. G. (July 16th, 1906), "A Clinical Picture of Relapsing Fever." *Journal of Tropical Medicine and Hygiene*, Vol. IX.

Spirochætes and Spirochætosis— *continued*

The following are the conclusions of Oppenheimer,[1] from work performed on *S. obermeieri* in New York. They are interesting as comparing the American parasite with *S. duttoni* and *S. carteri*:—

1. The New York *Spirochæta obermeieri* cannot yet, as has been attempted, be separated from the African spirochæte, upon the following grounds: (1) the length of its stay in the peripheral blood of the rat; (2) the number of relapses in the rat; (3) the lack of figure 8 and circular forms; (4) the absence of several transverse breaks; for the length of stay in the peripheral blood probably varies with the method of passage, relapses are an uncertain quantity, since it is, perhaps, not positively established that they occur at all, figure 8 forms and circles and finally several division zones exist in the New York spirillum as well as in *S. duttoni* and in the spirillum of Bombay.

2. As far as our work is concerned, the parasite merely holds its own *in vitro*; we cannot say that we had a culture.

3. It is not unlikely that the tangles and interwindings, seen during attempts at cultivation on artificial media, are due to the concentration or coagulation of the blood on standing, or the interwindings to fusion or conjugation.

4. The *Spirochæte obermeieri* probably increases by transverse fission and fragmentation.

5. We have seen no evidence of sporulation (no spore stain was used) or of a cycle of development, unless the particularly slender forms, short forms, "bands," and interwindings be considered such evidence. The only host studied was the rat.

6. The variations in the description of the motility are in all likelihood due to differences in the conditions under which the parasites are watched. As observed by us, its motility is almost precisely like that described by Hoffmann for *Spirochæta pallida*.

7. Perhaps the undulations that pass over the organism are merely an appearance, and the spiral is in truth rigid except for the lateral swayings.

8. The indication of an undulating membrane in the hanging-drop is the only sign of a definite structure which we have seen, except a deeply stained grain (granules). The absence of a complicated structure, the apparent multiplication by transverse division and fragmentation, the rapidity of multiplication, the length of viability outside of the body, and the persistence of the spiral form in death, point to a bacterium; whereas the flexibility of the parasite, the indication of an undulating membrane, the inability to cultivate the organism on artificial media, and the death at incubator temperature suggest that the New York *Spirochæta obermeieri* may be protozoan.

According to Schaudinn, *Spirochæta pallida* is not a spirochæte and not a spirillum, but a treponema. It is not a spirochæte, because of the permanency of its coils, because of its terminal cilium, and because it has not more or less blunted ends. It is not a spirillum, because its spirals are flexible, because it has a single cilium instead of a terminal tuft, and because it apparently divides longitudinally. The New York *Spirillum obermeieri* is certainly flexible and has permanent coils. If against our better judgment we grant in addition that each of its two ends represents a cilium and that the parasite divides longitudinally, then the New York *Spirochæta obermeieri* must be classed with *Spirochæta pallida* as a treponema.

Sprue. One has never heard of genuine sprue arising in the Sudan, but I understand that cases of somewhat intractable diarrhœa occur, so that some papers on this interesting condition may be noted. Moreover, the disease has been reported from some parts of tropical Africa, as Cantlie[2] records in a paper, wherein he also points out that it may attack old residents in the tropics or sub-tropics long after they have permanently taken up residence in Britain. He mentions also that sprue is associated with a diminution in size—an atrophy and atony—of the liver, which would appear more physiological than pathological in character. He believes the disease to be due to fermentative changes of micro-organismal or parasitic origin set up in the alimentary tracts of those who have been long resident in hot climates and are therefore debilitated. No definite organism has, however, been isolated.

The appearance of thrush in the mouth is a fatal sign (but *vide infra*). Cantlie has never seen a patient recover from sprue after thrush appeared. He condemns the milk treatment and commends the "meat" treatment, which he describes fully as follows:—

(1) The patient is put to bed, at any rate for a few days; (2) a wet pack is applied—a towel, or double layer of flannel large enough to reach from nipples to groins, and sufficiently wide to reach half-way round either loin—is wrung out in warm water and laid upon the abdomen. Round the body a large bath towel is wrapped, pulled tight and fixed by safety pins. The wet pack is kept on for two hours at a time, and is to be applied morning and evening; (3) the patient (unless very ill) is given 5 oz. pounded beef, lightly cooked, for breakfast, luncheon and dinner, with salt to taste, unless the mouth soreness prohibits its use. Every two hours the patient is given jellied beef-tea, beef-jelly, calves'-foot jelly, a plain jelly, all home-made; the jelly is given also during the night should the patient wake; (4) dr. 1½ castor oil every morning for first three mornings; (5) gr. 3

[1] Oppenheimer, A. (1906), "Laboratory Notes on Spirochæta Obermeieri found in New York." Collected Studies from the Research Laboratory Department of Health, New York, Vol. II.

[2] Cantlie, J. (November 2nd, 1905), "Sprue." *British Medical Journal*, p. 1282, Vol. II.

Sprue—continued

santonin morning and evening for three days. The effect of this treatment is that the stools at once become "bilious"; the bile, which seems to have been pent up, owing to previous "milk treatment," flows with great freedom when meat is given, and at first the stools are copious, loose, and as many as two or three daily. By the third day, in all probability, no stool is passed, and when on the fourth day the motion is seen, it is not infrequently fairly solid, fæcal and deeply bile-stained.

The diet may now (the fourth day) be increased, a poached egg is added to the beef at breakfast and dinner; pounded chicken may be given at the mid-day meal, instead of the beef. On the sixth day the meat or chicken may be finely minced—thrice passed through the mincing machine—in place of being pounded. By the eighth day the patient may have a cut off a joint, the undercut of a piece of roast beef being the best.

As soon as the stools are solid, or fairly solid, add vegetables to the diet—stewed celery, stewed sea-kale, or vegetable marrow; pulled and baked bread—thin slices of bread kept in a hot oven for twenty minutes until dry and crisp. The patient's diet is now fairly varied, and additional food can be tried—as judgment directs.

Rice-tea is made by roasting some rice in the oven until brown; then placing two tablespoonfuls of it in a jug and pouring over it one pint of boiling water and allowing the decoction to stand for fifteen minutes, when the rice is strained off and the "tea" used as a drink. China tea is made by taking the finest procurable, placing half a teaspoonful in a strainer over a breakfast cup and pouring boiling water over the leaves. Either of these teas, or each used alternately, may be given fifteen minutes after finishing food—never with the meat, of course.

The meat treatment, Cantlie holds, will not fail if carried out early and systematically. It goes well with the strawberry treatment.

In a discussion, Galloway differed from Cantlie on the subject of milk, and advocated the asepticisation of the bowel by means of calomel. This author has an exhaustive paper[1] on the subject, distinguishing an acute gastric (native) type and a chronic enteric type of sprue. Of intestinal antiseptics, apart from calomel, he recommends chinosol in sugar-coated "tabloids" of 5 grains each, thrice daily at first, but reduced even to one a day as the case improves. The remarks on dietetics appear to be sound and of value. In another paper[2] he shows how infection may take place by prolonged and intimate contiguity, proof of the organismal nature of the complaint.

Begg advised the exhibition of crude santonin, and Hartigan[3] prefers cyllin given in the form of intestinal palatinoids—3 m. cyllin in each. Younge[4] states he has known at least one case with thrush recover under treatment with pepsin, which, in the form especially of malto-pepsin, he has seen produce much benefit. It is given after meals in 5-grain doses.

Recently Cantlie[5] has had surprisingly good results in all cases of sprue by giving ipecacuanha in the same way as in dysentery. Its effects are most marked in advanced cases in which (*a*) the tongue is ulcerated and may have been extremely tender for months; (*b*) the stools are bulky and frothy; and (*c*) an increase of temperature occurs towards evening. The drug is given daily in 20-grain doses for two, three, or more days, and stopped when the stools become thin, brownish in appearance and devoid of odour (an ipecacuanha stool). Cantlie now advocates "fast-days" for patients on the meat treatment, *i.e.* days when milk alone is given as an "alterative" for twenty-four hours.

Begg[6] gives the complications found in chronic sprue, and which may prevent patients deriving full benefit from his santonin treatment. These are: (1) Pancreatitis; (2) Chronic appendicitis; (3) Involvement of liver and gall bladder with resulting jaundice; (4) Diabetes; (5) Pernicious anæmia. Mayo Robson[7] draws attention to the frequency of chronic pancreatitis in Europeans returning from tropical countries diagnosed as cases of sprue. He thinks that diseases of the pancreas are frequently unrecognised in the tropics. The same fact is recorded by Cammidge,[8] who mentions the special "pancreatic" reaction

[1] Galloway, D. G. (October 16th, 1905), "The Treatment of Sprue." *Journal of Tropical Medicine and Hygiene*, Vol. VIII.

[2] Galloway, D. G. (October 2nd, 1905), "Some Clinical Notes on the Etiology of Sprue." *Journal of Tropical Medicine and Hygiene*, Vol. VIII.

[3] Hartigan, W. (March 1st, 1905), "The Use of Cyllin in Sprue." *Journal of Tropical Medicine and Hygiene*, Vol. VIII.

[4] Younge, G. (December, 1905), "Pepsin in Sprue and Hill Diarrhœa." *British Medical Journal*, Vol. II.

[5] Cantlie, J. (September 2nd, 1907), "Ipecacuanha in Sprue." *Journal of Tropical Medicine and Hygiene*, Vol. X.

[6] Begg, C., *ibid.*, "Complications Found in Chronic Cases of Sprue." *Ibid.*

[7] Mayo Robson, A. W. (July 27th, 1907), "A Note on Interstitial Pancreatitis in its Relations to Sprue." *British Medical Journal*, Vol. II.

[8] Cammidge, P. J. (July 2nd, 1907). *Journal of Tropical Medicine and Hygiene*, p. 293, Vol. X.

Sprue—*continued*

in urine which indicates active degenerative changes in the pancreas (for details, see *British Medical Journal*, May 19th, 1906), and which may be employed to differentiate these cases from sprue.

Staining. There is no end to the production of new staining methods, and a large volume might be written on this subject alone, even if stains for blood only were considered. A few odd notes may, however, be useful.

One often has blood films sent in, which, owing to their age and their long exposure to heat, cannot be got to stain well by any of the Romanowsky methods. The red blood corpuscles do not take on the proper eosin hue, but appear green or greenish-blue, while the leucocytes at the same time often stain faintly. One had much trouble with these old films until Dr. Daniels, of London, kindly gave one the following method :—

Treat the slide *before staining* (and this is the special point) with a mixture of absolute alcohol one ounce and three to five drops of glacial acid for a few minutes. Wash in distilled water, then stain in the usual manner. Occasionally this procedure fails, but as a rule it succeeds admirably, and has proved of great service.

The best staining methods for the amœba of dysentery are not, as a rule, well described in English text-books, therefore a paper by Hart[1] on this subject may be quoted :—

Film preparations to demonstrate *Amœba coli* are, as a rule, generally found not to give satisfactory results. This is no doubt due to the comparatively large size and delicate structure of the organism.

By mixing in a test-tube a large volume of the infected material (either pus from a hepatic abscess or mucus from dysenteric stools) with a dilute logwood stain, and collecting the resulting dyed matter in glycerin, quite good results may be obtained.

The following are details of the method :—

STAINING SOLUTION

Hæmatein,	3 grains
Potassium alum,	1 grain
Acetic acid, B.P. (33 per cent.),	1 minim
Alcohol,	80 minims
Distilled water,	to 1 fluid ounce

To be used diluted with about four times its volume of distilled water.

1. Fill a test-tube to two-thirds of its capacity with the diluted logwood stain; add the infected material to the extent of about half-an-inch and diffuse by shaking gently for a few minutes. Any coarse particles are removed by straining through gauze and rejected. After straining for three hours the stained cells will be found to have settled to the bottom and the supernatant fluid can be poured off.

2. The colour of the stained structures is now to be developed by nearly filling up the tube with tap-water or a weak solution of lithium carbonate (half a grain to the ounce). Glycerin, sufficient to form a layer of, say, three-quarters of an inch at the bottom, is then carefully added to the tube. It is now set aside to allow the stained material to settle down into the glycerin. Deposition will have taken place in six or seven hours.

3. The supernatant fluid is poured off and the stained material in the glycerin transferred to slides and cover-glasses cemented on.

When the amœbæ are to be mounted in Canada balsam the procedure is somewhat different and not quite so simple. Glycerin must of course be omitted.

The water having been poured off from the stained material, dilute alcohol is added and the tube is set aside for an hour or two. The deposit is then collected on a filter paper and drained.

The filter paper with its contents is then folded up to form a packet, tied with a thread, and suspended in absolute alcohol to dehydrate. When dehydration is considered complete, transfer the packet to some clearing agent and again to a fresh supply of the clearing agent. (Rectified coal-tar naphtha answers the purpose admirably and is inexpensive.)

The cleared preparation, after draining, can then be mounted in Canada balsam in the usual way.

Another method is that used by Dr. Wenyon, and which, though a little tedious, produces excellent results. It is here given as shortly as possible :—

Fixing fluid = Saturated sublimate, 2 parts.
Alcohol absolute, 1 part.

Fæces spread out by needle on cover-slips. Without drying, drop slip, film-side down, on the fixing fluid, best warmed to about 60° C. After a few minutes, reverse film and allow to sink. Fix for twenty to thirty minutes. Wash in distilled water for five minutes, and then place in 70 per cent. alcohol to which a few drops of Gram's Iodine has been added. Leave in this at least two hours (for a day if one likes). Transfer to distilled water and stain with Heidenhain's Iron Hæmatoxylin, Delafield's Hæmatoxylin or Borax Carmine.

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[1] Hart, T. (December 15th, 1903), "The Preparation of Permanent Stained Specimens of Amœba Coli." *Journal of Tropical Medicine and Hygiene*, Vol. VI.

Staining—*continued*

Iron Hæmatoxylin. Iron alum water solution 2½ to 4 per cent., six hours or longer. Wash for about one minute in distilled water. Then in Heidenhain's Hæmatoxylin about twelve hours. Wash in water and differentiate under microscope in a watch-glass with ½ per cent. solution of iron alum.

Delafield's Hæmatoxylin. Must be used dilute about four or five drops to a Petri dish of distilled water. Stain film in this for twenty-four to forty-eight hours.

Borax Carmine. One part to about five or six parts of 70 per cent. alcohol. Differentiate for about half to one hour in 2 per cent. acid (HCl) alcohol.

After staining, films are passed through alcohol and xylol, and mounted in balsam.

Films must never be allowed to dry during the whole process.

Fixing in bulk. If fæces are solid, mix with saline and pour into test-tube to depth of about one inch. Fill up with fixing fluid (as above, or Flemming's) and gently shake to mix well. Allow fæces to settle; this takes about one hour. Remove supernatant fluid and replace by water. Allow to settle and repeat, washing twice. Then add 70 per cent. alcohol and iodine and leave for several hours. Remove fluid and add stain—either dilute Delafield's Hæmatoxylin or Borax Carmine (after Borax Carmine add acid alcohol). Take up into absolute alcohol by this sedimentation method. In another test-tube place at bottom clove oil, then layer of mixture of clove oil and absolute alcohol, above this the fæces in absolute alcohol. Fæces will gradually sink to the bottom, when fluid can be removed and fresh clove oil added. Examine in clove oil.

Plimmer[1]* has a new method for demonstrating trypanosomes so as to show the finest cytological details. The following is the technique employed :—

(1) Expose a cover-slip to the vapour of osmic acid (1 per cent.), 1 c.c. glacial acetic acid 3-5 drops for 2 minutes; (2) place a drop of fresh blood on one corner of the slip and expose again to the vapour for 30 seconds; (3) spread the film carefully and expose again for 15–30 seconds to the vapour until the surface appear no longer moist; (4) place slip in absolute alcohol for 10 minutes; (5) immerse slip in faintly rose-coloured solution of permanganate of potash for 1 minute (2-3 drops of 1 per cent. sol. to 50 c.c. H_2O); (6) wash in water for 5 minutes; (7) stain in the following modified Romanowsky, made by mixing just before use—azur 1 (1 per cent.) 1 c.c.; eosin. A_1 (1–1000) 2 c.c., H_2O 8 c.c. for 15–30 minutes; (8) wash; (9) differentiate in orange tannin 30 seconds; (12) alcohol xylol (2-3) two or three changes; (13) xylol; and mount.

Instead of 7 to 13, any other method of staining can be used, according to what structures it is desired particularly to show.

Huisman[2]* studied 17 different methods for staining blood films, and found Jenner's best and simplest, but he has improved upon it.

The stain he uses is a mixture of equal parts of a 1·175 per cent. solution of solid azur-blue in pure absolute methyl alcohol, and a 0·825 per cent. solution of eosin BA (Hochst) in the same medium. He stains for two minutes without previously fixing the film. By this method the nuclei appear a violet-blue, while the basophile protoplasm takes a light blue stain which gives a good differentiation from the rest of the preparation. The red corpuscles are rose-coloured. The neutrophile granules are rose-violet, violet, or violet-blue; basophile granules are blue; the metachromatic basophile granules are violet-red, and the oxyphile granules are red. The author also observes that some of the lymphocytes exhibit, when stained by this formula, evidence of a fine blue or metachromatic granulation.

Urtubey[3]* has invented what he calls a simple, rapid and certain method of preparing the Leishman stain. Those who are accustomed to use the "soloids," and to be satisfied with them, are not likely to resort to this new method, but a reference is given for any who may wish to try it. It is simpler than the usual complicated process. A new Gram's method, in which both the stain and the Gram's liquid are altered, gentian-violet being superseded by methyl-violet 6.B. or methyl-violet B.N., and the Gram's fluid by that of Unna, which yields nascent iodine, is given by Lœffler.[4]* It is said to yield excellent results even in unpractised hands.

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[1] Plimmer, G., "Demonstrating Trypanosomata." *Proceedings of Royal Society*, Ser. B. LXXIX., pp. 95-102.

[2] Huisman, A. (1906), "The Staining of Blood Films." *Méd. et Hyg.*, No. 4. Quoted in *British Medical Journal* Epitome, July 21st, 1906, p. 12.

[3] Urtubey, A. (February 28th, 1907), "Note sur un procédé simple, rapide et sûr pour preparer le colorant de Romanowsky-Leishman." Quoted in *Bull. de l'Institut Pasteur*, Vol. V.

[4] Lœffler, F. (August 2nd, 1906), "Zur Gramschen Färbungsmethode." *Deut. Med. Woch.* Quoted in *Bull. de l'Institut Pasteur*, October 30th, 1906, Vol. IV.

* Article not consulted in the original.

Staining— *continued*

Herman[1] recommends a new staining method for tubercle bacilli, applicable both to smears and sections. The staining bath is composed of a mordant consisting of a 10 per cent. solution of carbonate of ammonia in distilled water and of a stain, namely, 3 per cent. solution of crystal violet in ethylic alcohol at 95° C.

One part of the stain is added to three parts of the mordant immediately before use and the solution well mixed. Decoloration is effected by a 10 per cent. mixture of nitric acid in ethylic alcohol at 95° C.

MacNeal[2]* has introduced a rapid and simple method of staining *Spirochæta pallida*, with the following solution: methyl violet 0·25, medicinal methylene blue 0·10, eosin 0·20, methylic alcohol pur. 100. Dissolve, aiding solution by warming. The film to be stained is covered with the undiluted solution for 45 to 60 seconds. It is then immersed for one or two minutes in 10 c.c. of a solution of sodium carbonate of a strength of 1 part in 20,000. Rinse in distilled water, dry and examine with an immersion lens. The spirochætes are intensely stained, and retain their colour if mounted in rectified Canada balsam.

Syphilis. This is a subject of great importance in the Sudan, where the disease is rife, occurring both in mild and severe forms. Its prevalence has already been noted and commented upon in the First Report of these Laboratories; but, though of special interest, it is utterly impossible to review more than the merest fraction of the vast number of papers which have accumulated on the subject since the discovery of the *Spirochæta pallida*. Indeed it is only the resemblance of syphilis to spirochætosis and trypanosomiasis, which justifies the brief notice accorded it here.

Mott,[3] in an interesting paper, draws attention to the similarity existing in several respects between syphilis and trypanosomiasis, saying:—

> As showing the analogy with trypanosome infections, I may mention that it was observed by Lingard that in the *mal du coit* of horses scarification of the plaques, which come out in successive crops—commencing forty days after infection—yielded blood and serum containing numbers of trypanosomes, whereas they were found with difficulty in the peripheral blood. Nattan-Larier and Tanon likewise found in the fluid obtained by scarification of the erythmatous spots in a white person suffering from trypanosomiasis, numbers of trypanosomes, whilst none could be found in the peripheral blood stream.
>
> In both these diseases the lymphatic glands generally become enlarged, and in sleeping sickness puncture of the swollen glands and examination of the fluid reveals trypanosomes; so also Metchnikoff was able to demonstrate the spirochætes in the enlarged glands of the infected chimpanzee. It therefore follows that there is this analogy between the virus of syphilis and the before-mentioned trypanosome diseases—that the protozöon multiplies in the lymph with much greater readiness than it does in the blood; this may be due to the presence in the blood plasma of lysins, similar to the bacteriolysins or hæmolysins. Moreover, there is so close a similarity in the reaction of the fixed tissue elements to the presence of the trypanosomes or spirochætes, that either the organisms themselves or the toxins generated by them give rise to a similar hyperplasia characterised by proliferation of the nuclei of the fixed connective tissue cells forming the embryonic cells or lymphocytes, and proliferation of the endothelial cells, forming plasma cells or epithelioid cells. In trypanosome diseases, however, there is no proliferation of the subendothelial cells of the arteries, leading to endarteritis, vascular occlusion, and necrobiosis of the central cells, as in a gumma. It is, moreover, remarkable that, unlike bacterial infection, trypanosome affections and syphilis are, if uncomplicated by secondary microbal affections, unattended by polymorphonuclear invasion.

Leishman[4] reviews the papers read on the etiology of syphilis at the Berlin Congress of Hygiene, 1907. He quotes Hoffmann as saying that:—

> For demonstration of the spirochæte, Giemsa's stain was best for fresh films and Levaditi's silver method for sections; but he pointed out that it was also possible to stain the parasite in sections by Giemsa's stain, and to demonstrate them in films by the silver method, if proper precautions as to technique were followed. For diagnosis, he thought the best method was staining by Giemsa, combined with a search for the unstained spirochætes with dark-ground illumination. The spirochætes are found chiefly in the connective tissue, in the lymphatic vessels, and in the walls of the blood vessels; occasionally they are to be met with in leucocytes, hepatic cells and other tissue cells. The conditions found in the blood are not, apparently, favourable to the development of the spirochæte, possibly because it is an anærobe.

Hoffmann believes the spirochæte to be undoubtedly the cause of syphilis, and states that it holds the same diagnostic importance for syphilis as the *B. tuberculosis* for tubercle. Zabolony concurs in this view, and he pointed out that the spirochætes are agglutinated by the addition of serum derived from syphilitic patients.

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[1] Herman, M. (January 25th, 1908), "Sur la coloration du bacille tuberculeux." *Ann. de l'Institut Pasteur*, Vol. XXII.

[2] MacNeal, W. J. (February 16th, 1907). *Journal of the American Medical Association.*

[3] Mott, F. W. (January 4th, 1908), "An Address on some recent Developments in our Knowledge of Syphilis in Relation to Diseases of the Nervous System." *British Medical Journal*, Vol. I.

[4] Leishman, W. B. (April, 1908), "The International Congress of Hygiene, Berlin, September, 1907." *Journal of the Royal Army Medical Corps*, Vol. X.

* Article not consulted in the original.

Metschnikoff dealt with prophylaxis, especially as regards the use of his preventive ointment, the formula for which is:— **Syphilis**—*continued*

Calomel,	33	grammes
Pure lanoline,	67	,,
Vaseline,	10	,,

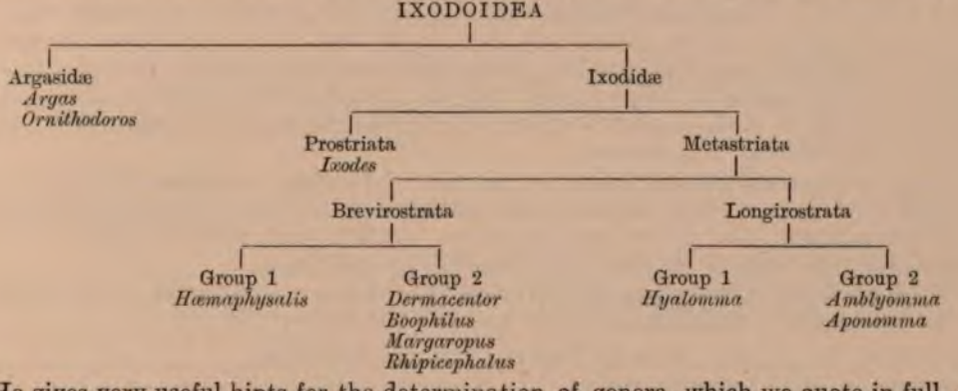
If properly and carefully prepared on this basis, and if applied within a few hours of exposure to the risk of infection, Metschnikoff considers the above ointment as an absolute safeguard, but as it is certainly useless at a later period, he has continued his researches in the hope of finding some preventive treatment, which would be successful when applied some days after exposure. He tried atoxyl treatment both in the case of men and monkeys, but though it possesses a certain value, the toxicity of the drug is against it, and Metschnikoff aims at finding some other efficacious but less toxic form of arsenic.

Possibly the 'Kharsin' and 'Orsudan' of Messrs. Burroughs Wellcome & Co., which have been tried in syphilis by Ward,[1] may meet the case. (*See* special paper on "Trypanosomiasis," Third Report.)

Neisser,[2*] who experimented on apes, concluded that it was impossible to obtain a serum which will produce immunity. He successfully inoculated apes which had been infected twice before and been twice cured. He believes a radical cure of the disease is possible by treatment with mercuric iodide and also atoxyl, and is convinced that the serum diagnosis of syphilis is useful and affords a reliable test of complete cure. In the *Medical Annual* for 1907, a good *résumé* of some of the more important recent work will be found, including staining methods for *S. pallida*, the results of the examination of tertiary lesions, and the contagiousness of gumma.

Ticks. A useful monograph, especially to those working in Africa, is that by Dönitz,[3] who gives synoptic tables of genera and species, as well as a general biological account of these insects.

The generally accepted classification of ticks is that of Neumann, but with this Warburton[4] declares himself to be dissatisfied and proposes, in lieu of it, the following:—



He gives very useful hints for the determination of genera, which we quote in full, and reference should be made to his attached diagrammatical figures which will greatly facilitate recognition:—

Males and slightly distended females should be selected for examination if possible.

The rostrum or capitulum and scutum are generally sufficient to indicate the genus, and these should be examined first, the conclusion being verified by reference to other structures. It is soon seen that there is a long type and a short type of rostrum.

If the rostrum is long, the genus is either *Ixodes*, *Hyalomma*, *Amblyomma*, or *Aponomma*.

If the palps are narrow at the base, leaving a gap between them and the hypostome, it is probably *Ixodes*. Verify by looking for the anal groove in front of the anus, the absence of eyes and festoons, and the circular or oval peritremes.

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[1] Ward, W. A. (April, 1908), "Atoxyl and its Value in the Treatment of Syphilis." *Ibid.*

[2] Neisser, A. Quoted in *British Medical Journal*, January 11th, 1908, Vol. I.

[3] Dönitz, W. (Leipzig, 1907). "Die Zecken."

[4] Warburton, C. (December 11th, 1907). "Notes on Ticks." *Journal of Economic Biology*, Vol. II., No. 3.

[*] Article not consulted in the original.

Ticks— *continued*

Palps of fairly uniform width throughout indicate one of the other genera.

If there is no great difference in the length of the palpal joints, and spherical eyes are present, it is probably *Hyalomma*. If a male, it may be determined immediately by the four anal plates and two additional posterior chitinous joints.

If the second palpal joint is much the longest, it is either *Amblyomma* or *Aponomma*, the first having eyes, the second, not. Moreover, the tick will probably be ornate, and an ornate long-rostrumed tick from a domestic animal is sure to be *Amblyomma*.

If the rostrum is of the short type, its shape leaves little doubt of the genus.

If the width across the base of the palps is considerably greater than that of the transversely rectangular rostral base the genus is *Hæmaphysalis*. Verify by the absence of eyes.

Dermacentor may have a somewhat rectangular rostral base, and more or less bulging palps, but eyes are present, and the ticks are generally ornate. If a male, note the fourth coxæ, much longer than the others.

In *Rhipicephalus* and *Boophilus* the rostral base is hexagonal, being pointed laterally. Distinguish by the extremely short-ridged palps and circular peritremes in *Boophilus*, and the moderate unridged palps and comma-shaped peritremes, of *Rhipicephalus*.

Notes on the diagnosis of species follow, and he points out the great variability that exists and the presence in many species of "high males" and "low males." The latter are small and poorly characterised specimens, the former large and with their characteristic points greatly emphasised. He says that the extremes of series would certainly be taken for different species, but numerous intermediate forms connect them. From a list of the principal species attacking domestic animals, with notes on their distribution, we pick out those which are found in Africa, and refer the reader to Mr. King's paper on "Sudanese Ticks" (Third Report).

Hæmaphysalis punctata.—Chiefly on sheep.
Hæmaphysalis leachi.—In South Africa. On the dog.
Margaropus (single species only).—In South Africa. On the horse.
Rhipicephalus sanguineus.—On all domestic animals. (The dog tick of the Sudan.)
" *bursa*.—On all domestic animals.
" *simus*.—On all domestic animals.
" *evertsi*.—On all domestic animals.
" *capensis*.—In South Africa. On cattle.
" *appendiculatus*.—In South Africa. On cattle.
Hyalomma ægyptium.—On all domestic animals.
Amblyomma hebræum.—In South Africa. Chiefly on cattle.
" *variegatum*.—On cattle and sheep. (A variety is common in the Southern Sudan.)

Massey[1] gives a long list of ticks collected by him from various animals on the Congo-Zambesi water-shed, and identified for him by Neumann and Nuttall. Amongst these we note a few not given in Warburton's list, *i.e. Rhipicephalus lunulatus*, *R. supertritus*, *R. attenuatus* and *R. gladiger*, all new species found on the horse; also a variety of *Margaropus annulatus (decoloratus)*, from the ox.

Some of these and several other new species of Ixodidæ, have been recently described at length by Neumann,[2] who has also described[3] two new African species from bovines under the names *Rhipicephalus duttoni* and *R. longus*. In the same paper he gives a table of differentiation of males of the various species of *Rhipicephalus*.

Nuttall[4] begins his Harben Lectures for 1908 with a consideration of the Ixodoidea, and we transcribe a useful table which he has compiled:—

LIST OF DISEASE-BEARING IXODOIDEA

Redwater in cattle is transmitted by:—
Boophilus annulatus.
B. dugesi (australis).
B. decoloratus.
Ixodes ricinus.
Rhipicephalus capensis.
Hæmaphysalis punctata (in experiments by Stockman). Suspected are *Rhipicephalus evertsi* and *Hyalomma ægyptium*, since Koch states he observed the development of *Piroplasma* in these species.

Rhodesian fever in cattle is transmitted by:—
Rhipicephalus appendiculatus.
Rhipicephalus simus.

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[1] Massey, A. Y. (March 2nd, 1908), "Some Ticks of Central Africa." *Journal of Tropical Medicine and Hygiene*, Vol. XI.

[2] Neumann, L. G. (March 20th, 1908), "Notes sur les Ixodidés." *Arch. de Parasit.*, t. XII., No. 1.

[3] Neumann, L. G. (February 1, 1907), "Description of two new Species of African Ticks." *Annals of Tropical Medicine and Parasitology*, Ser. T.M., Vol. I., No. 1.

[4] Nuttall, G. H. F. (July, 1908), "The Ixodoidea, or Ticks." *Journal of the Royal Institute of Public Health*, Vol. XVI, No. 7.

Ticks—
continued

Redwater in sheep is transmitted by:—
Rhipicephalus bursa.

Malignant jaundice in dogs is transmitted by:—
Hæmaphysalis leachi.
Rhipicephalus sanguineus.

Biliary fever in horses is transmitted by:—
Rhipicephalus evertsi.

Heartwater in goats and sheep is transmitted by:—
Amblyomma hebræum.

Rocky Mountain spotted fever in man is transmitted by:—
Dermacentor occidentalis.

Spirochætosis in fowls is transmitted by:—
Argas persicus.
(also by *A. reflexus* and *Ornithodoros moubata*, as has been experimentally proved).

Spirochætosis in cattle is transmitted by:—
Boophilus decoloratus.

Spirochætosis in man (African relapsing fever) is transmitted by:—
Ornithodoros moubata.

An interesting account is given of the preventive measures in force against *Boophilus* in the United States.

Lounsbury,[1] in a paper on ticks and East Coast fever, while showing that the usual carrier of the disease is *R. appendiculatus*, incriminates four other species of the same genus, one of which, *R. nitens* (the Shiny-Brown Tick) has not yet been mentioned in this review. The others are the Red Tick (*R. evertsi*); the Capensis Tick (*R. capensis*); and the Black Pitted Tick (*R. simus*). He gives a description of the different species which, with the exception of *R. evertsi*, possess a similar life-history. While they resemble each other in appearance the members of this group are easily distinguished from the various other ticks that frequent cattle. The same author, in speaking of cattle dip for ticks, says:—(1) Arsenic is the principal agent which acts on the ticks in these dips; (2) That only the preparations containing the largest percentage of arsenic, that is, only those that were estimated to contain one pound of arsenic to 30 gallons of water, ever destroyed the ticks; and in most cases even these strongest mixtures failed to prevent a few females from maturing. Mostly, however, such females die without laying eggs, and nearly all the eggs laid, failed to hatch.

Mayo[2]* states that in Cuba a solution of Cebadilla seed in spirit has been found very efficient, as has the so-called Cuban dip, which is a modification of dips used in Australia and South Africa, and is made up as follows:—

Arsenious acid,	8 lb.
Soda carbonate cryst.,	24 lb.
Yellow soap,	24 lb.
Pine-tar,	1 gallon.
Water,	500 gallons.

Dissolve the arsenic in 20 gallons or more of water by boiling for 30 or 40 minutes. When dissolved, add 100 gallons of water. Dissolve the soap and soda in 20 gallons of boiling water, first shaving the soap, and while boiling add the pine-tar in a thin stream and stir until it is dissolved. Mix this with the arsenical solution and add sufficient water to make 500 gallons.

This solution kills the ticks and does not irritate the cattle, but it does cause some irritation to the thin skin of the arms of men who work in the solution for more than two days.

It may be used as a hand-dressing or with a spray-pump, and gives excellent results.

It is interesting to note that in the Sudan the natives employ, with some success, a solution of henna as a dressing for fowls infested with *Argasidæ*.

Manson[3] gives a short account of the history of *Ornithodoros moubata*, and Newstead[4] has also described it. Wellman adds a note and correction on the length of time which elapses between impregnation and egg-laying. This proved to be nearly two months (fifty-seven days). The females are known to live four months after ovipositing.

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[1] Lounsbury, C. P. (May, 1906). *Cape of Good Hope Agricultural Journal*, Vol. XXVIII., No. 5.

[2] Mayo, N. S. (June, 1906), "Dips for Cattle Ticks." *American Veterinary Review*, Vol. XXX. Quoted in *Journal of Tropical Veterinary Science*, October, 1906, p. 454.

[3] Manson, Sir P. (London, 1907). *Tropical Diseases*, 4th Edition.

[4] Newstead, R. (August 15th, 1905), "On the Pathogenic Ticks concerned in the Distribution of Disease in Man, with Special Reference to the Differential Characters of Ornithodoros Moubata." *Journal of Tropical Medicine*, Vol. VIII.

* Article not consulted in the original.

Ticks— *continued*

To anyone studying ticks, Christophers'[1] monograph on their anatomy and histology is indispensable. He has a short chapter on ticks as transmitters of disease, and gives the following as being human parasites:—

Argas persicus	(Fischer)
Argas tholozani	(Lab. et Meg.)
Ornithodoros moubata	(Murray)
Ornithodoros savignyi	(Andouin)
Ornithodoros savignyi var. *cæca*	(Neumann)

The last is probably identical with *O. moubata*. *O. savignyi* is the human tick found in the Northern Sudan. Other ticks which attack man are the Mexican species, *Argas turicata* and *A. megnini*, and the pigeon tick, *A. reflexus*. It will be noticed that all these belong to the *Argasidæ*. In Massey's list (*loc. cit.*), however, one notes a human tick, *Rhipicentor bicornis* (Nuttall and Warburton), belonging to a new genus. It, and several other new ticks, have been recently described and figured by Nuttall and Warburton.[2]

Christophers deals with the bionomics, external anatomy and internal structure of ticks, taking *Ornithodoros* as one type and *Rhipicephalus* and *Hyalomma* as the other. The method he recommends for dissection is, one can say from personal experience, not very difficult, and, thanks to the excellent illustrations he gives, it is comparatively easy to follow out the somewhat complex internal organs.

Tropical Medicine. Under this comprehensive title one proposes to discuss a few papers on very varied subjects which cannot well be considered under any of the other headings, and yet deal with matters of very considerable interest and importance to the student of Tropical Medicine.

We note first a paper by Carnegie Brown[3] on degeneration of the myocardium in hot climates, in which he says:—

> A somewhat unfamiliar result of residence in the Tropics, but one which is by no means infrequent, is a peculiar form of degeneration of the cardiac muscle. Clinically and pathologically the condition is of much interest, for the evidences of myocardial change are apt to be misinterpreted, and sudden and unanticipated death occurs not infrequently after an apparently trivial illness.
>
> The patient is usually a man who has been some years in the Tropics; he is confident that he is organically sound, and though he confesses he is not well, he is sure that there is little the matter. In answer to leading questions he says he is tired, yet sleepless; he is disinclined for exertion, mentally or bodily; he yawns unaccountably; he has flushings of the face, tingling and itching in the extremities; perspires more readily and copiously than is his habit; and he has a vague feeling of uneasiness and apprehension. There is no faintness, no dyspnœa, no cough, no pre-cardiac oppression. Palpitation is absent, or it is slight, and felt only after exertion. There is, perhaps, some indigestion, and he has remarked that the feeling of weariness comes on coincidently with discomfort in the stomach. He is thirsty, and wants more than his usual quantity of stimulant. He is not depressed, but he worries unnecessarily about his affairs, and is kept awake at nights by a stream of thought and indefinite anxiety. The pupils are large, and not actively responsive to volitional or reflex stimuli. The pulse is irregular, but it is full, soft and deliberate; the vasomotor system is inactive, and there is no hardening of the arteries. The skin is moister and more lax than usual. The temperature is normal; the tongue is clean, and gives no indication of any morbid condition; the urine is scanty and loaded with urates, though otherwise normal. The rhythm of the natural cardiac cycle is unimpaired, but the heart's action is somewhat hesitating, and an occasional flutter or tremor may be detected at the apex; the impulse is softer, perhaps more diffuse, than normal, but it is not displaced. The clearness of note in the sounds is diminished; they seem a little distant, and the first is somewhat weak. Little further aid is to be obtained from the stethoscope; there are no adventitious murmurs, and examination of the pulmonary area and conus arterious gives no indication of deficient or defective blood.

He notes that the condition is apt to be considered as nervous or imaginary, and yet a startling and tragic termination may ensue, possibly as a result of transitory fever or indisposition.

> Contrasted with the commoner forms of myocardial decay the salient features of the disease are the absence of fainting fits, the inconsiderable impairment of the circulation, the vagueness of the physical signs, and the fact that the syncope usually supervenes in the absence of unwonted exertion. It is perhaps only a coincidence, but one the importance of which will not be overlooked, that death frequently occurs at night and after the administration of a soporific.

The post mortem appearances are given and may be summed up as those of "soft heart," so far as the macroscopical appearances are concerned. Its histo-pathology, however, is quite different from that of the special condition which bears this term. The appearance rather resembles cloudy swelling, and is evidently due to impaired nutrition.

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[1] Christophers, S. R. (1906), "The Anatomy and Histology of Ticks." *Scientific Memoirs of the Government of India*, No. 23.

[2] Nuttall, G. H. F., and Warburton, C. (1907). *Proceedings of the Cambridge Philosophical Society*, Vol. XIV., Pt. IV.

[3] Carnegie Brown, W. (June 23rd, 1906), "Degeneration of the Myocardium in Hot Climates." *British Medical Journal*, Vol. I.

The disease is one of the imprudently strenuous life, and an exciting cause is over-indulgence in hard exercise.

Timely treatment may arrest the destructive process and may possibly even remedy the condition to some extent. The cardiac muscle is starved, not for lack of nourishment but from want of ability to absorb the nutritive material which is around and about it in superfluity. If its metabolic capacity and nutrition can be restored, so, certainly, will its contractibility and ability for work. To this end, rest in bed is the first essential, and diet must, in consequence, be limited. General massage is necessary to maintain and restore muscle function and assimilation. Heart stimulants—digitalis, ether, strychnine—are to be avoided; strong cigars, than which there is no more powerful cardiac depressant, must be prohibited. If the patient is a moderate drinker, alcohol should be stopped; if he is a free liver and stimulants appear indispensable, consideration is necessary. But in deciding the point, which is very important, it should be borne in mind—and this is specially true of the Tropics—that the sudden withdrawal of alcohol and tobacco from a patient thoroughly habituated to their use may induce such deep dissatisfaction as to seriously prejudice all assimilative processes. As to drugs, opium is of special value. In the Tropics, where heat exhaustion is in daily evidence, its efficacy is more generally appreciated than in colder climates. There can be no question of the beneficial action of opium in the extreme fatigue that is induced by continued and severe physical effort in hot climates, and in the dyscrasias that result from over-exertion. In no disorder in the Tropics is this more apparent than in degeneration of the myocardium; apart from its sedative effect, opium acts with promptitude and certainty as a restorative of cardiac tissue; and in such a condition it may be safely and usefully continued for long periods without other medicine than an occasional laxative. All other hypnotics should be discarded; some of them are dangerous, others may be so. With suitable treatment, the prognosis of this form of myocardial degeneration is by no means bad, and if taken in hand during their earlier stages most cases will do well.

Cantlie,[1] in a useful paper, discusses tropical ailments as they are met with in Britain, in the course of which he mentions that a man (even a medical man) may suffer from malaria whilst resident in the Tropics and yet be unaware of the fact. He mentions a malarial neuritis which may supervene on chronic malaria in Britain. He believes that it is not the cold but the hot weather in Britain which is to be dreaded by the victim of chronic malaria. He has found 10 minims of oil of turpentine, repeated every two hours until five doses were given, useful in checking hæmoglobinuria occurring at home in persons who have had black-water before, or who develop it for the first time in Britain. He cautions against mistaking the presence of intestinal parasites for dysentery or sprue, especially in cases where loose stools alternate with normal motions. In such cases the effects of santonin should be noted. In women suffering from any kind of intestinal flux, the pelvic organs should be examined and any gynæcological trouble remedied.

True tropical dysentery in Britain is, he thinks, best treated by ipecacuanha. A good section is that on "Tropical Liver." Cantlie points out that one must not expect to find the "livery" tropical subject with an enlarged liver. It is just as frequently diminished in size. When the liver is small (he says) give it work to do, give the patient meat; when it is large and tender and there is increased temperature, take the strain off it and give milk.

As useful drugs, he mentions chloride of ammonium, rhubarb and soda, one of the purgative waters, and occasional doses of calomel or ipecacuanha are indicated.

Castellani[2] has described as occurring in Ceylon an "Endemic Funiculitis," being an acute, inflammatory condition of the spinal cord, which is apt to lead to general septicæmia and death, unless prompt operative measures (orchiotomy) are taken. He describes the pathology, and states that he has found a diplo-streptococcus in all cases. He is inclined to believe that this organism is merely a secondary agent, playing a rôle only in the suppurative stage of the malady.

Military medical officers may refer with advantage to a review[3] of a paper by Steuber on European troops in the Tropics. Unfortunately the original article is in German, and there is nothing to show that it has been translated into French or English, but it seems to possess much of interest and alludes to the last Sudan campaign.

Bousfield[4] has described in the Sudan an intractable, slow-spreading, destructive ulcer of the penis. He saw three cases occurring in the Sudanese. The ulcers, which were practically painless and apparently non-syphilitic, are described as having no covering slough and exuding a serous fluid which at times was slightly purulent. Smears revealed nothing special—nothing like Ducrey's bacillus of soft sore was found, and there were no spirochætes. Various kinds of treatment failed until, in one case, oil of turpentine was tried with happy results, apparently due to the medication.

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[1] Cantlie, J. (June 22nd, 1907), "Tropical Ailments as they are met with in Britain." *British Medical Journal*, Vol. I.

[2] Castellani, A. (July 4th, 1908), "Endemic Funiculitis." *Lancet*, Vol. II.

[3] Steuber, — (August 10th, 1907), "European Troops in the Tropics." *British Medical Journal*, Vol. II.

[4] Bousfield, L. (September 14th, 1907), "Observations from the Soudan." *Lancet*, Vol. II.

Tropical Medicine— *continued*

Attention may be directed to a paper on microscopical technique as applied in Tropical Medicine, by Langeron.[1] A great deal of useful information is given, especially perhaps with reference to the staining, mounting and preservation of parasitic worms.

Mention must also be made of a new protozoal parasite, *Histoplasma capsulata*, found by Darling[2]* in a case from Panama. The parasite, which somewhat resembles the Leishman-Donovan body, produces in the lungs a condition of pseudo-tuberculosis, and in the liver, spleen and lymph nodes areas of focal necrosis. In the lung the majority of the bodies were found in the interior of the epithilial cells lining the alveoli, which, detached and distended, were often united in masses simulating tubercles. In the liver the parasites invade the hepatic cells and the endothelial cells. In the spleen and bone marrow the majority are free in the plasma. They may be found in the mononuclear leucocytes, as is the case with those in the lymphatic glands.

A second case of this curious infection has been encountered.

Trypanosomiasis. On no subject connected with tropical medicine has such a mass of literature recently accumulated as that dealing with the pathogenic and non-pathogenic trypanosomes. The question, so far as human beings are concerned, has already been discussed under Sleeping Sickness, while as regards the Sudan it is the subject of a special article (Third Report).

Hence it will not here be necessary to enter into the matter very fully especially as Nabarro's[3] translation of Laveran and Mesnil's classical work presents English readers with a recent and very complete review of the whole question. Nearly every week, however, something new is discovered, so that some reference to it is necessary.

Fraser,[4] in the Malay States, succeeded in definitely proving that *Tabanidæ* were capable of transmitting trypanosomes. This is of interest as, so far as one knows, this had only been hitherto definitely proved in the case of *Atylotus nemoralis* and *At. tomentosus* in Algeria. That this family of biting flies must frequently be concerned is, however, certain from experience in India, Abyssinia, the Sinai peninsula and other regions where the tsetse is wholly unknown. *Hippoboscidæ*, of course, may sometimes be effective as in the case of *H. rufipes* and *T. theileri* in South Africa. As to the implication of Stomoxys and other blood-sucking insects, Nabarro's work must be consulted.

The question of big game and "fly" will be found discussed in *The Asian* for November 2nd, 1907, the article being reviewed in the *Journal of Tropical Veterinary Science* for May, 1908.

Papers on Surra, in India, and in Indo-China, by Holmes and Schein respectively, the former entering into the results of treatment with atoxyl, tartar-emetic, perchloride of mercury, etc., appear in the *Journal of Tropical Veterinary Science* for May, 1908, and are likely to interest those working in these countries.

França[5]* has recorded a curious observation :—

Some blood from a frog, doubly infected by *T. costatum* and *T. rotatorium*, was left between slide and cover, sealed with paraffin. Four days after the author was astonished to find in place of the trypanosomes a large number of small, very mobile flagellates, resembling spirochætes and herpetomonads. The nucleus stains deeply, and the blepharoplast is placed anteriorly, and gives origin to a very distinct flagellum. On repeating the observation, the author finds that after two days a sort of dissociation of the costæ of *T. costatum* takes place, resulting in fusiform protoplasmic masses, more or less active, which ultimately separate, leaving behind a residuum of amorphous protoplasm. A flagellum is then developed at one end, by means of which they move about in the blood, though the other ends are still adherent. When stained, these forms show a small nucleus and a blepharoplast at the flagellate end.

Besides these, distinctly culturable round forms were met with, and along with them trypanosomes preparing to divide. In these latter there may be distinguished a series of nuclei and blepharoplasts placed in the projecting ribs on the surface of the parasite, the projection gradually becoming pinched off and separating. Lastly, in preparations where the blood was, to begin with, strongly oxygenated, small trypanosomes, with a simple undulating membrane, which he regards as intermediate forms, were met with.

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[1] Langeron, M. (March 20th, 1908), "Technique Microscopique Appliquée à la Médècine Coloniale." *Arch. de Parasit.*, Vol. XII., No. 1.

[2] Darling, S. T. (April 28th, 1906). *Journal of the American Medical Association.*

[3] Nabarro, D. (London, 1907), "Trypanosomes and Trypanosomiasis."

[4] Fraser, H. (1906), Rep. Inst. Med. Res. Fed. Malay States.

[5] França, C. (November 1st, 1907). Quoted in *Journal of Tropical Medicine and Hygiene*, p. 360, Vol. X.

* Article not consulted in the original.

Trypanosomiasis—*continued*

Martin[1] has described in French Guinea a curious form of trypanosome, *T. boueti*, in a lizard, which, to judge from the drawings of fresh preparations, must be difficult to recognise as a trypanosome. It is a mass of protoplasm with amœboid movements and characteristic movement of the undulating membrane. The flagellum does not appear to be longer than the body. Motion is restricted. The nucleus and centrosome are difficult to distinguish. In stained specimens the nucleus is seen to be spindle-shaped and more or less curved, while the centrosome is found to be situated at one of the ends of the spindle. It would seem that the undulating membrane is the most characteristic portion of this parasite, which is altogether peculiar.

Marchoux and Salimbeni[2] have found in a tree-frog of Petropolis another strange trypanosome, not of the *T. rotatorium* type. The name suggested for it is *T. borelli*, and it is not unlike the *T. boueti* just mentioned. Its nucleus is spindle-shaped and has the centrosome situated at its anterior extremity.

Brief reference to these, and to most of the other trypanosomes described up to the end of 1907, will be found in Sambon's special paper in the last edition of Manson's "Tropical Diseases." Attention has recently been drawn to what is called the axial filament in trypanosomes. This was described by Robertson[3]* in *T. brucei* as a non-chromatic line, staining a violet blue by Romanowsky running along the long axis of the parasite from the centrosome to the anterior extremity of the body. Moore and Breinl[4] working with wet films and employing special fixing and staining solutions describe, as has been already mentioned under "Sleeping Sickness" *(page 175)*, an intra-nuclear centrosome and various granules near the broad end of the body of the parasite, which they propose to call extra-nuclear centrosomes (blepharoplasts). It is from one of these that the flagellum springs. In *T. gambiense*, at certain periods of infection, they also describe what is probably the axial filament of Robertson which seems first to have been noted by Prowazek. They picture it as a relatively-thick stainable band proceeding from the extra-nuclear centrosome. It is most readily stained by iron hæmatoxylin, less readily by various aniline colours. It grows from the extra-nuclear centrosome, not along the surface of the animal, but down the interior of the cell towards the nucleus. It is fully twice as thick as the flagellum, and appears near the periods when the greatest number of parasites are present in the blood.

It may reach or even pass the nucleus; or it may become coiled upon itself. It seems, however, to become undoubtedly connected up with the nucleus and is evidence of some sort of interaction taking place between the extra-nuclear centrosome and the nucleus. Such a phenomenon, say Moore and Breinl, occurs only among animals in which no evidence of nuclear division or cell division is evident. During the whole development of the stainable band the nucleus remains in a condition of complete repose. It is suggested that the metamorphoses connected with the appearance of the "black line" is an attempt on the part of the trypanosomes to become sexually differentiated, but this attempt is not completed, the cells reverting to their primary condition, in which case the process could be regarded as an example of a special form of parthenogenesis. In this connection it is noted that at the time of the formation of the black line only a portion of the extra-nuclear centrosome unites with the nucleus. Swellengrebel[5]* has found the axial filament in *T. brucei*, *gambiense* and *equinum*. The intra-nuclear portion of the axial filament appears to be the centre for the formation of granules which afterwards leave the nucleus in the line of the filament and then disperse themselves throughout the cytoplasm. These granules are specially abundant in parasites which are degenerating and consists of volutine.

Salvin-Moore, Breinl and Hindle[6] have recently applied their special methods to the study of the life-cycle of *T. lewisi* which closely resembles those of *T. gambiense* and

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[1] Martin, G. (April 13th, 1907), "Sur un Trypanosome de Saurien." *C. R. Soc. Biol.*, t. LXII. Quoted in *Journal of Tropical Veterinary Science*, November, 1907.

[2] Marchoux, A., and Salimbeni, E. (*ibid.*), "Un Trypanosome Nouveau chez une Hyla Voisine de H. Lateristriga." *Ibid.*

[3] Robertson, M. (1906). *Proceedings of the Royal Society, Edinburgh*, Vol. XVI.

[4] Moore, J. E., and Breinl, A. (December 9th, 1907), "The Cytology of the Trypanosomes." *Annals of Tropical Medicine and Parasitology*, Series T.M., Vol. I., No. 3.

[5] Swellengrebel, N. H. (January 18th, 1908), "La Volutine chez les Trypanosomes." *C. R. Soc. Biol.*, t. LXIV. Quoted in *Bull. de l'Institut Pasteur*, April 15th, 1908.

[6] Salvin-Moore, J. E., Breinl, A., and Hindle, E. (July 1st, 1907), "The Life-History of Trypanosoma Lewisi." *Annals of Tropical Medicine and Parasitology*, Series T. M., Vol. II., No. 3.

* Article not consulted in the original.

Trypanosomiasis— *continued*

T. equiperdum as described by them. In *T. lewisi*, however, the inter-action between the extra-nuclear centrosome and the nucleus is followed by the formation of characteristic multi-nucleated masses. These again are succeeded by the assumption of the peculiar morphology of the round flagellated "latent body" which eventually passes into the trypanosome form. One point specially noted is that whereas in the pathogenetic forms, *T. gambiense* and *T. equiperdum*, the phases of the life-cycles as they appear among the trypanosomes do so nearly simultaneously among all the parasites existing in the blood at a particular time, and thus mark successively the stages of the infection; in the non-pathogenetic form, *T. lewisi*, all the stages of the life-cycle may be present and represented by different parasites which are found in the blood at the same time.

The authors note that in the case of all three trypanosomes mentioned they have quite failed to find any evidence of the so-called males, females and other forms described by Schaudinn.

An interesting paper, and one of very considerable importance, is that by Novy, MacNeal and Torrey[1] on the trypanosomes of mosquitoes and other insects. Some of their conclusions are here tabulated:—

1. Of the "wild" mosquitoes examined by us approximately 15 per cent. in one year, and about 5 per cent. in that following, were found to be infected with flagellates belonging to two easily differentiated types. *Crithidia* and *Herpetomonas*. The percentage of infections is influenced largely by local and seasonal conditions.

2. By the cultivation method it has been possible to isolate two of these flagellates, namely *Crithidia fasciculata* and *T. culicis*; and it has been shown that the plate method is applicable for the separation of trypanosomes from the accompanying bacteria and yeasts.

3. The organisms obtained *in vitro* correspond to those observed *in vivo*, and hence the intestinal types represent true cultural forms.

4. The two types are common in other insects, and, instead of being classed as distinct genera, they should be placed under the trypanosomes.

5. The mosquito flagellates are not stages of intracellular organisms, but are probably parasites peculiar to the insects.

6. The inoculation of available experimental animals with the *Crithidia* and *Herpetomonas* has given negative results.

7. The trypanosomes which are at times present in ticks are not developmental forms of *Piroplasma*. It has already been shown that the trypanosomes in tsetse flies are not related to *T. gambiense* or *T. Brucei*.

8. The possibility of the trypanosomes of birds and other vertebrates developing in the gut of insects, while not excluded, has not been demonstrated. In the mosquito, *T. lewisi* and *T. brucei* lose their infectiousness, more or less rapidly, but the enfeebled organism may survive in the gut for 36-48 hours. The conditions in the digestive tube of insects is not as favourable as in the test-tube, and this fact goes to show that insect carriers, such as tsetses, are mere passive hosts.

Morax[2] has a paper on ocular manifestation in trypanosomiasis, and concludes that the lesions, on account of their frequency and character, are of great interest. The appearance of a non-ulcerated interstitial keratitis in animals may, in many cases, lead us to suspect the existence of trypanosomiasis.

This interstitial keratitis is provoked by the multiplication of trypanosomes in the interlamellar spaces of the cornea. The proliferation of the parasite leads to an infiltration of leucocytes, then a vascular development. These lesions may cause complete disorganisation of the cornea. They may disappear, leaving very slight traces after them. This occurrence is most common in animals which show a marked resistance to infection by trypanosomata. This was the case in the goat attacked by Dourine or Nagana. Dogs, on the contrary, die whilst the cornea is still completely opaque.

The interstitial keratitis occurring in dogs inoculated with the Sudan trypanosome of mules was described in the Second Report of these Laboratories.

It is of interest to note that recently Yakimoff and Schiller[3] reported that rabbits, guinea pigs, dogs, white and grey rats, but not white mice, can be infected by feeding with *Trypanosoma lewisi* and the trypanosomes of El-Debab (a disease of dromedaries in Algeria), Nagana, Surra and Dourine. Experiments with the trypanosome of Mal-de-Caderas yielded negative results.

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[1] Novy, F. G., MacNeal, W. J., Torrey, H. N. (April 10th, 1907), "The Trypanosomes of Mosquitoes and other Insects." *Journal of Infectious Diseases* (Chicago), Vol. IV., p. 269.

[2] Morax, V. (January 25th, 1907), "Manifestations oculaires au cours de Trypanosomiases," *Ann. de l'Institut Pasteur*, Vol. XX.

[3] Yakimoff, W. L., and Schiller, N. (1907), "Zur Trypanosomen infektion durch die Schleimhaut des Verdaungstraktes," *Cent. f. Bakt. Abt I.*, Bd. XLIII., H. 7.

Nierenstein[1] has conducted observations on the acidity and alkalinity of the blood in trypanosome infection, and concludes :— **Trypanosomiasis—** *continued*

i. It is evident that in experimental trypanosomiasis infection (*T. brucei* and *T. equiperdum*), the acidity of the blood increases.

ii. The increase of the acidity is probably due to the production of amido-acids through or by the trypanosomes.

iii. It is possible that the increase of acidity might be of assistance in the diagnosis of a typical case of trypanosomiasis, where the parasites have disappeared for some length of time from the blood circulation.

iv. These experiments suggest that in trypanosome treatment effort should be made to neutralise the increased acidity of the blood, as this might prove of additional assistance in making the blood a less favourable medium for their development.

Laveran and Thiroux[2] discuss the rôle of the spleen in trypanosomiasis, and conclude :—

1. That when observations are made under good conditions the trypanosomes found in the spleen during life or immediately after death present the same appearance as those encountered in the general circulation.

2. Splenic extract has no trypanolytic properties *in vitro*.

3. In animals on whom splenectomy has been performed the evolution of trypanosomiasis is not sensibly modified.

4. In trypanosomiasis, as in malaria, the spleen doubtless contributes to free the circulation from the débris of hæmatozoa following the trypanolytic crises, but its rôle seems limited to this function.

This leads one to speak of the causes of trypanolytic crises and the relapses that follow them. Massaglia[3]*

worked with guinea pigs infected with surra, which presented, during the evolution of the malady, a certain number of disappearances of the trypanosomes, followed by renewed proliferation. He first established that, although normal guinea pig serum has but slight action on the trypanosomes of mice, that of infected guinea pigs has marked trypanolytic power if taken just before the crisis, and acts very powerfully during or after the crisis. If these sera are put up to the trypanosomes of guinea pigs, the result is but little less intense if the parasites be taken just before the crisis, but they prove almost refractory after it. Heating for fifteen minutes to 65° C. entirely destroys the trypanolytic power of the serum.

In vivo guinea pig serum taken during the crisis has no curative action and but little preventive power.

Rodet and Vallet[4]* took the blood of a dog with experimental nagana daily from the date of infection to that of death, defibrinated and centrifuged it, and then put it up to rat trypanosomes taken at the beginning of infection. At the outset the trypanolytic power of the serum is nil, and only begins to appear two days before the first crisis, but during it and after, it is very strong. The agglutinating power reaches its maximum immediately before the crisis. The dog's own trypanosomes proved less sensitive to the action of the serum than those of rats. Heating even to 55° C. more or less destroys the power of the serum.

From these data the authors conclude that the trypanolytic crises are due to the action of the serum on the trypanosomes of the circulating blood, and are in no way a consequence of the crises.

Some papers on preventive and curative methods may be cited. Certain of Panse's[5]* experiments in connection with immunisation against Nagana are interesting. He found the Koch-Schilling's method of attenuation by passage through various species of animals unsatisfactory on account of the persistence of trypanosomes in the immunised animals. Injections of bile from infected animals made before, or simultaneously with, a virulent inoculation brought about a mild attack, followed by considerable immunity, one of the experimental oxen resisting five inoculations with a very virulent virus. Two oxen treated preventively for four months with injections of blood containing dead trypanosomes were also found to be immune. Out of two animals treated with a mixture of blood and bile, one died after six months on inoculation with the virus, while the other was found to be immune.

Lastly, the author tested the protective value of the serum of the immunised animals. When mixed with trypanosomes it produced a mild attack in one dog out of several

[1] Nierenstein, M. (July 1st, 1907), "Observations on the Acidity and Alkalinity of the Blood in Trypanosome Infections." *Annals of Tropical Medicine and Parasitology*, Series T. M., Vol. II., No. 3.

[2] Laveran, A., and Thiroux, A. (August 25th, 1907), "Sur le rôle de la rate dans les Trypanosomiases." *Ann. de l'Institut Pasteur*, Vol. XXI.

[3] Massaglia, A., "The causes of Trypanolitic Crises, and the Relapses that follow them." *C. R. Acad. Sc.*, t. CXLIV. Quoted in *Journal of Tropical Medicine and Hygiene*, May 1st, 1908.

[4] Rodet, A., and Vallet, G. *Ibid.*

[5] Panse, —, "Attempts at Immunisation against Nagana in German East Africa." *Deutsch Colonialblatt*, No. 7, p. 8. Quoted in *Journal of Tropical Medicine and Hygiene*, April 15th, 1908.

* Article not consulted in the original.

Trypanosomiasis— *continued*

inoculated, and one ox survived, while the check animal died in five days. It appears, therefore, that there is some probability of the desired result being attained.

Goebel[1] has a paper on the preventive and curative power of human serum on the infection of nagana. He finds that it has no opsonic or cytotropic power in regard to trypanosomes and appears in no way to act by a combination of alexin and sensitising bodies. Its preventive power has been verified for the mouse.

Amongst the latest suggestions as regards treatment, not mentioned in Nabarro's treatise, is that of Castellani[2] with reference to the use of quinine cacodylate. He says :—

1. Quinine cacodylate shows a well-marked trypanocide action in monkeys experimentally inoculated with a strain of trypanosome closely resembling *T. evansi*.

2. Mercury perchloride improves the general condition of the inoculated monkeys, and frequently causes the temperature to subside to normal. Given alone, however, its trypanocide action is not very marked. In patients affected with sleeping sickness, it would seem from the experiments made by Low and myself in 1903 that mercury perchloride given by intravenous injection induces a fall in the temperature, but the improvement is only temporary.

3. I suggest that quinine cacodylate alone, or, better, in combination with intravenous injections of perchloride of mercury, should be tried in sleeping sickness, using large doses. If it should be found that quinine cacodylate has a marked trypanocide action also on *T. gambiense*, the advantage of this drug over other preparations would be its powerful action on the malaria parasites. This would be an appreciable advantage, as in the experience of many observers, among whom are Low and myself, a large number of sleeping sickness patients are suffering also from chronic malaria.

4. Quinine cacodylate, besides its trypanocide properties and its action on Laveran's parasites, has apparently some action on other protozoa, as, in my experience, its use is beneficial in kala-azar and yaws.

Laveran and Thiroux[3] recommend the administration of atoxyl together with orpiment, and report cures in experimental animals infected with *T. gambiense*.

Mesnil and Nicolle[4] bring their results with atoxyl and the colour Ph. (Afridol-violet), in the case of monkeys inoculated with *T. gambiense*, up to date in a paper wherein they affirm that they have definitely cured six of the animals by atoxyl alone, four by alternating atoxyl with Ph., two by Ph. alone, at first, and then associated, at the conclusion of treatment, with one or two doses of atoxyl.

Browning,[5] a disciple of Ehrlich, reviews the whole question of chemo-therapy in trypanosome infection. The article, which is very erudite, requires careful study. One would only note here that the work seems to show that differences in the immunity-reaction are not a sufficient basis for the classification of different species of trypanosomes.

Reference may be made to a special article in the *Lancet* for January 11th, 1908, on the treatment of trypanosomiasis, which details all the recent work, including the various combinations adopted by Plimmer and Thomson who used the lactate, succinimide and sozoiodol of mercury, and also iodipin along with atoxyl.

Another important paper for reference is that of Weber,[6]* who details every kind of treatment which has been tried, dealing with immunity, active and passive, and sero-therapy, the use of extracts of organs and of their secretions, the employment of bacteria and their products, and chemical and physical methods.

Special references and reviews will be found in the following numbers of the *Bulletin de l'Institut Pasteur*, July 30th, 1907, September 30th, 1907, and January 15th, 1908.

Lœffler and Russ,[7]* working with Nagana, have revived the treatment with arsenious acid, and have been able to cure severely affected animals (guinea pigs, rats and rabbits) in a relatively short time, and to confer on healthy animals the power of resisting repeated

[1] Goebel, O. (November 25th, 1907), "Pouvoir preventif et pouvoir curatif du Sérum humain dans l'infection due au Trypanosome du Nagana." *Ann. de l'Institut Pasteur*, Vol. XXI.

[2] Castellani, A. (February 29th, 1908), "Note on the Treatment of Experimental Trypanosomiasis." *British Medical Journal*, Vol. I.

[3] Laveran, A., and Thiroux, A. (February 25th, 1908), "Recherches sur le traitement des Trypanosomiasis." *Ann. de l'Institut Pasteur*, Vol. XXII.

[4] Mesnil, F., and Nicolle, M. (December 25th, 1907), "Traitement des infections experimentales à Trypanosoma Gambiense." *Ann. de l'Institut Pasteur*, Vol. XXII.

[5] Browning, C. H. (January, 1908), "Chemo-Therapy in Trypanosome Infections: an Experimental Study." *Journal of Pathology and Bacteriology*, Vol. XII.

[6] Weber, H. (1907). *Zeitsch. für Exp. Path. und Ther.*, Bd. IV.

[7] Lœffler, F., and Russ, V. K. (August 22nd, 1907). *Deut. Med. Woch.*

* Article not consulted in the original.

Trypanosomiasis—*continued*

inoculations with the parasite. The drug can be given by the mouth, intravenously or intra-peritoneally. The solution of arsenic should be prepared by boiling 1 gm. of arsenious acid with 10 c.c. of normal caustic soda and subsequently adding 10 c.c. of normal saline solution. The lethal dose per kilo of body weight is greater by about one-third than the curative dose. Both doses differ for the different species of animals, but with care there is no fear of poisoning. The arsenious acid acts directly upon the trypanosomes, not through the medium of the leucocytes. The hyperleucocytosis which occurs is a secondary phenomenon consequent on the destruction of the trypanosomes. It would seem that the arsenic must be present in an adequate degree of concentration in the blood and tissue juices if a complete effect is to be obtained. Otherwise, though the trypanosomes may disappear from the circulation, they are not wholly destroyed, and relapse occurs. *In vitro* a dilution of 1 in 200,000 is efficient.

Magalhæs,[1] following up Kopke's work, has shown that certain drugs given by the mouth or subcutaneous injection do not pass into the cerebro-spinal fluid, and so do not affect any trypanosomes that may happen to be there. Iodine, potassium iodide and methylene blue were employed both by the mouth and intramuscularly, but though later they were found abundantly in the urine, they never penetrated the meninges. Apparently also atoxyl, which has a real action on certain of the symptoms of the malady—notably on the fever and on the disappearance of the trypanosomes from the blood and the glands—does not penetrate, because, according to the author, the trypanosomes always persist in the cerebro-spinal fluid. The conclusion to be drawn from this, therefore, is that, in order to attack them effectively in this position the drugs used must be introduced under the arachnoid directly.

Reference may be made to a paper by Moore, Nierenstein and Todd,[2] dealing with the resistance shown by the parasites (notably *T. brucei*, *T. gambiense* and *T. dimorphon*) to drugs, and the changes in virulence of the strains after they have escaped the drug action and have reappeared in the blood. Atoxyl and acetylated atoxyl were used.

Tsetse Flies. Of considerable importance and interest is the discovery by Carter[3] of *Glossina tachinoides* (Westwood), in Southern Arabia, where it was found in several different districts, but in no great numbers. It is said not to depend for its existence on big game, because, excepting gazelle, nothing else frequents the belts of bush which it haunts. Natives stated that it bit goats, donkeys, horses, dogs and men, but did not attack camels or sheep.

Stuhlman has published an exhaustive monograph dealing chiefly with *Glossina fusca*, but *G. tachinoides* also receives some consideration. In a review[4]* of his paper the following points are noted:—

1. Males greatly preponderated in his collections, which is strange, because, in the case of pupæ, the proportion was found to be equal. 2. A species of cocco-bacillus and a pink yeast organism were found to be common in the digestive canal. 3. A hungry tsetse will absorb from 1·26 to 2·7 times its own weight of blood. 4. As regards reproduction, it was found that a female laid eight larvæ in three-and-a-half months, and that the pupa stage lasts from thirty to sixty-five days. Twice females not certainly fecundated gave birth to larvæ. 5. As regards the influence of external conditions an average temperature of 23° to 26° C., with a maximum of 36° to 37° C., and a minimum of 10° to 12° C., is necessary, combined with a degree of relative humidity ranging from 66 to 83 per cent. 6. In nature it was found that from 3 to 14 per cent. of *G. fusca* contain trypanosomes within the proboscis, while the digestive tube is infected in a much larger proportion. 7. Koch's description of the forms of trypanosome found is confirmed—undifferentiated forms being found in the hinder part of the intestine, elongated forms in the proventriculus and œsophagus, and small forms chiefly in the proboscis. 8. Experimentally, infection is most easily effected on the occasion of the fly's first meal after escaping from the pupal case. 9. The evidence is rather against the possibility of hereditary transmission. 10. What may have been a conjugation of parasites was once observed in the proventriculus, and conjugation is said to be a necessary prelude to the appearance of small forms in the proboscis, which constitute the agents of infection of vertebrates. Attempts to infect the latter by means of injections of emulsion of the fluid obtained from the proventriculus uniformly failed.

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[1] Magalhæs, J. de (December 31st, 1906), "Troubles cerebelleux et bulbaires dans la Maladie du Sommeil." *Arch. de Hyg. et Path. Exot.* (Lisbon), Vol. I., fasc. 2.

[2] Moore, B., Nierenstein, M., and Todd, J. L. (July 1st, 1907), "Notes on the Effects of Therapeutic Agents on Trypanosomes in respect to (*a*) Acquired Resistance of the Parasites to the Drug, and (*b*) Changes in Virulence of the Strains after Escape from the Drug." *Annals of Tropical Medicine and Parasitology*, Series T. M., Vol. II., No. 3.

[3] Carter, R. M. (December 17th, 1906), "Tsetse Fly in Arabia." *British Medical Journal*, Vol. II.

[4] Stuhlman, F. (March 2nd, 1908), "Contributions to our Knowledge of the Tsetse Flies." *Journal of Tropical Medicine and Hygiene*, Vol. XI.; also in *Bull. de l'Institut Pasteur* (December 30th, 1907).

* Article not consulted in the original.

Tsetse Flies *—continued*

Minchin[1] reported fully on the anatomy of *G. palpalis*, in a well-illustrated paper, followed by a preliminary article on the relation of this fly to *T. gambiense*. This work is now so well known to all interested in the subject that there is little need to discuss it in detail. Suffice to say that Minchin and his fellow-observers, Gray and Tulloch decided that the trypanosomes found in freshly-caught tsetse flies, and named by Novy *T. grayi* and *T. tullochii*, have nothing to do with sleeping sickness, and are not developmental stages of *T. gambiense*. They were not able to determine on what vertebrate host, if any, these trypanosomes are parasitic, but indicate two possible sources for them (*a*) some of the numerous animals, water birds, crocodiles, hippopotami, etc., upon which the fly feeds; (*b*) these trypanosomes may be parasites of the fly itself, like the *Herpetomonas* of the domestic fly, or, one may add, of the Nile Seroot.

Their observations showed that *T. gambiense* itself actually does die out in the tsetse fly after the third day. It was only found in the mid-gut of the fly.

In the same publication, Minchin describes the occurrence of encystation in *T. grayi*, the form produced resembling what is found in *Herpetomonas* infections. Dealing with the significance of this process, he says:—

> It seems to me in the highest degree improbable, indeed, I may say impossible, that a tsetse-fly would ever infect itself by sucking up cysts dropped by another fly, or that a parasite which had to depend on this method or dissemination could maintain its existence in the tsetse fly. The only possible destiny I can imagine for these cysts is to be swallowed accidentally by some vertebrate, the (as yet unknown) host of *Trypanosoma grayi*, in order to germinate in its digestive tract, to pass thence into the blood, and to be taken up again with the blood by the tsetse fly. A cycle of this type is as yet unknown, but there are abundant analogies for all parts of it. In the first place, it is a common thing for animals to have protozoan parasites in the gut, which they take up in the encysted condition after they have been dropped by another individual. Without multiplying instances unnecessarily, I may point out that Schaudinn proved the infection of *Amœba coli* to originate in this way, and that it is a common human parasite in regions where sanitation has not advanced beyond the primitive condition of *epandage par terre*. In the second place, there are many instances among Sporozoa of cysts germinating in the intestine and liberating motile forms which then pass through the wall of the gut into other organs of the body.
>
> In a former communication by my colleagues, Lieutenants Gray and Tulloch, and myself, we were able to confirm Bruce's results as to the existence of direct mechanical infection by means of the tsetse-fly, which if it stabs its proboscis first into an infected animal and then soon after into a healthy one, can infect the latter. We were not able to demonstrate, however, what I may term cyclical infection, which at present has not been shown to exist. I suggest that there are two possible modes of cyclical infection, in the dissemination of protozoan blood-parasites by biting insects generally. In one method, which I may term *inoculative*, the parasite, after going through developmental changes in the insect, passes back again into a second vertebrate host through the proboscis, as in the case of malaria transmitted by a mosquito. In the other method, which I propose to term *contaminative*, the parasite taken up by the biting insect, after going through developmental changes within its gut, would pass out through the anus, and infect the vertebrate host by contaminating its food or drink. We have all of us (I speak for myself) been imbued hitherto with the idea that the cycle of the trypanosome in the tsetse fly must be of the inoculative type, and have failed to find it. I wish to suggest strongly to those working on the subject of trypanosome-infection the desirability of making experiments and observations to prove or disprove the existence, in the insect which disseminates the parasite, of a life-cycle which results in a contaminative infection of the vertebrate host.

His later work and conclusions[2]* have already received brief consideration under the heading "Sleeping Sickness" (*page* 174).

Tuberculosis. Such a wide subject cannot be fully discussed in a review like this, but allusion will be made to such points as may be of interest and importance.

Reference may be made to the absorption of tubercle bacilli by the skin. Nouri and Osman[3]* found that if guinea pigs were shaved in the inguinal region and then rubbed with absorbent cotton fouled with tuberculous sputum, the corresponding lymph glands enlarged and became swollen in eight to fifteen days, and the animals died in thirty to fifty days. This may explain to a certain extent the puzzling location of certain tubercular lesions which are met with in Mohammedan and other countries where the art of shaving is somewhat crudely performed, the victim being shaved with water and a blunt razor and the operator frequently adding his saliva as a soapy adjuvant!

As regards the presence of tubercle bacilli in the blood, Lüdke,[4] was able to isolate the tubercle bacillus from the blood of consumptive patients by withdrawing 5 c.c. to 10 c.c.

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[1] Minchin, E. A. (February, 1907), "Report on the Anatomy of the Tsetse Fly." *Report Sleeping Sickness Comm. Roy. Soc.*, No. VIII.

[2] Minchin, E. A. (March, 1908), "Investigations on the Development of Trypanosomes in Tsetse Flies and other Diptera." *Quarterly Journal of Microscopical Science*, New Series, No. 206, Vol. LII, pt. 2.

[3] Nouri, O., and Osman, "Absorption of Tubercle Bacilli by freshly shaven skin." *C. R. Soc. Biol.*, t. LIV., p. 308.

[4] Lüdke, H. (1906). *Wiener. Klin. Woch.*, No. 31, p. 949.

* Article not consulted in the original.

of blood from the median basilic vein and injecting it into the peritoneal cavity of guinea pigs. In three out of fourteen cases tubercle bacilli were thus found to be present in the circulating blood.

Reference may be made to rather an important observation from a prophylactic point of view, viz., the method of sedimenting and disinfecting sputum at the same time by means of hydroxyl. Sachs-Mücke[1] found that on the addition of hydroxyl to sputum, a stormy evolution of oxygen gas is produced and the gas bubbles tear and break up the tough solid sputum masses. He recommends the addition of equal parts of hydroxyl and 1 per cent. solution of perchloride of mercury to the sputum receptacles.

Thus, with a minimum amount of trouble and danger, the daily amount of sputum may be both disinfected and sedimented.

As regards the diagnosis of tuberculosis, the method of examining the sputum for tubercle bacilli has been frequently attended with the difficulty of obtaining a uniformly even smear, thick enough to present a fair sample of the particular specimen under examination, yet not so thick as to obscure the light.

Rickards[2] in a paper describes an apparatus by means of which all masses of sputum previously coagulated by 5 per cent. carbolic acid solution and all caseous particles are broken up rapidly and the sputum is rendered homogeneous throughout. This method of shaking up the sputum gave a gain of 2·8 per cent. of positive results in detecting the tubercle bacillus. Rickards found that, by the addition of a small amount of 10 per cent. aqueous solution of caustic soda previous to the shaking, digestion of the mucus took place more readily, rendering the smearing easier. He recommends a Babcock machine for sedimenting the sputum.

In connection with the diagnosis of the tubercle bacillus by the Ziehl-Neilsen method of staining, a very important recent paper by Dr. Hans Much[3] shows that this observer found, on making post mortem examinations of cattle infected with tubercle bacilli of known origin and ordinary character, and presenting typical nodules in the lung, that the most careful examination in certain cases may fail to reveal acid-fast forms, and this also in spite of the fact that inoculation experiments may demonstrate the tuberculous nature of the lesions and that from some of the cases tubercle bacilli may even be obtained by culture.

Similar results were observed in the case of the so-called "cold abscesses" occurring in the human subject. Much found by using the methods of Gram and Ziehl-Neilsen, that there were two forms of non-acid-fast tubercle bacilli; one a rod-shaped form, partly granular, and the other a granular form consisting of granules lying singly or clustered together into irregular groups. In using Gram's method the preparations were left in the gentian violet solution for 48 hours before decolorisation.

Further, he showed that by taking small pieces of lung tissue from a case where no ordinary tubercle bacilli were found, and incubating for varying periods in serum tubes placed in an incubator at 37° C., when stained by Gram only granules and rods were obtained; but after incubating for six days, bacilli staining by Ziehl-Neilsen's method were recognisable. In a paper on the same subject in the *Beitrage zur Klinik der Tuberculose*, Band VIII, Heft 1, page 85, Much suggests that the Ziehl-Neilsen's staining depends upon some other constituent of the bacillus than that upon which the Gram method rests.

Michaelides, in a paper in the same number of that journal, arrives at similar conclusions. He also states that there is a form of the tubercle bacillus which does not stain with Ziehl-Neilsen or with Gram, but which can be demonstrated by the Loeffler-Giemsa method of staining. These recent observations are evidently of considerable practical importance in diagnosis.

A valuable and recent aid to the diagnosis of tubercle was furnished by Calmette[4] in June, 1907, and is now known as the "Calmette Ophthalmo-reaction."

Calmette announced that if tuberculin be placed in the eye of a tuberculous subject a conjunctivitis is produced, whereas in a healthy subject there is no change.

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[1] Sachs-Mücke (October, 1906), "A method of Sedimenting Sputum by Hydrogen Peroxide." *Journal of Preventive Medicine.*

[2] Rickards (May, 1907), "Sputum shaking and Sedimenting Apparatus." *Journal of Infectious Diseases.*

[3] Much, H. (June 6th, 1908). *Berlin Klin. Woch.*

[4] Calmette, A. (August 30th, 1907), "Sur un nouveau procédé de diagnostic de la tuberculose chez l'homme par l'ophthalmo-réaction à la tuberculine." *Bull. de l'Institut Pasteur.*

Tuberculosis *—continued*

It is essential in employing this test that the eye to be tested should be healthy and free from abrasion. A 1 per cent. solution in sterile distilled water of a dried precipitate prepared by the addition of 95 per cent. alcohol to tuberculin is employed, but in some cases this was found to produce too violent a reaction, and now a 0·5 per cent. solution is in general use. If the patient be tuberculous, a reaction takes place which may begin in three to twenty hours after the application of the test. The maximum reaction may be reached in from eight to sixteen hours, the duration of the reaction may be from two to ten days. The inflammation may vary from a mild lachrymation with conjunctivitis and an almost imperceptible enlargement of the caruncle to a severe purulent conjunctivitis which may last for several days. There is, as a rule, no rise of temperature and no general malaise. Fortunately, no case has as yet been recorded in which the eye has been permanently damaged. The several reports show that the reaction bears no relation to the degree of activity of the tuberculous lesion nor to the extent of the lesion.

From the various results recorded, as regards the Calmette Ophthalmo-reaction, it cannot be regarded as an infallible index of the presence or absence of tubercle, although it must be considered as a valuable aid to diagnosis. Cases have been recorded in which a severe reaction has been obtained in apparently healthy men, and the reaction has failed in undoubtedly tuberculous individuals. In connection with this latter point, it is of interest to note that persons with healed tuberculous lesions fail to give this reaction, so that this test can, to a certain extent, be used as a criterion for determining when a tuberculous lesion is cured. Eyre, Wedd and Hertz,[1] in 138 cases tested, obtained a positive reaction in sixty-three cases and in the remainder no reaction occurred. The majority of the positive results were undoubtedly tuberculous. Lecky has published further statistics on the Tuberculin Ophthalmo-reaction, and his results show that in cases known to be tuberculous, 94·3 per cent. gave positive reaction; in cases probably tuberculous, 66·2 per cent. gave positive results, and in cases considered to be healthy or non-tuberculous, only 7·4 per cent. gave a positive reaction. These figures are of interest in showing, to a certain extent, what reliability can be placed on Calmette's Ophthalmo-reaction as an aid in the diagnosis of tubercle.

Harrison Butler[2] found this reaction of use in diagnosing whether tubercle was the cause of cerebral compression in two of his cases. Continental observers have found that the reaction cannot be obtained even in undoubtedly tuberculous cases during the last week of life, and this view was confirmed by Eyre and his co-workers in a case of tuberculous meningitis that died thirty hours after the test was applied. It only remains for time to show what an important part the Calmette Ophthalmo-reaction will play in the social struggle against tuberculosis.

Calmette's test shows that newly-born children of tuberculous mothers do not give the reaction, but in such children of 1 to 2 years of age, 3 per cent. give a positive result and the percentage increases rapidly with age. These observations are in accordance with those of Bang and Nocard, who state that tuberculosis in cattle is scarcely ever congenital. By repeating the test sufficiently often, Calmette suggests that the exact time at which tuberculosis attacks a child may be ascertained. The part played by family contagion and by the milk of tuberculous cows may be determined by studying the condition of the family and the food supply. If the test be applied periodically to the members of a family in whom tuberculosis is feared, the infection may be detected at an early period even before clinical signs develop themselves, and the necessary precautions as regards isolation and treatment may be begun at an early period of the disease. Further, Calmette's reaction would be of use in the examination of pupils seeking admission to schools and who are suspected of being tuberculous. In the Army and in the Navy the test could be put to a similar use.

Slatineance[3] made an interesting observation in connection with the appearance of the Calmette eye reaction after a subcutaneous inoculation with tuberculin. He states that a subcutaneous inoculation of tuberculin, after application of the Calmette test, produced a new specific reaction on a level with the Calmette test. This reaction was produced as well in the tuberculous as in healthy individuals. Its intensity was greater if the interval between the two operations was a short one. In tuberculous cases he obtained a

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[1] Eyre, J. W., Wedd, B. H., Hertz, A. F. (December 21st, 1907), "The Tuberculin Ophthalmo-reaction of Calmette." *Lancet*.

[2] Harrison Butler, T. (April 18th, 1908), "Calmette Ophthalmo-reaction." *British Medical Journal*.

[3] Slatineance, A. (August 30th, 1907), "Le vérial de l'oculo-réaction de Calmette par l'injection souscutanée de Tuberculine." *Bull. de l'Institut Pasteur*.

reaction after an interval of 31 days between the two operations, but in normal, healthy persons this reaction did not occur after an interval greater than eight days between the two operations.

The rôle that flies play in relation to the transmission of tuberculosis has been worked out by André[1]* of Lyons. He observed that flies which have had access to tuberculous sputum retain the tubercle bacilli in their digestive tubes for several days, and that the tubercle bacilli multiply there more rapidly than in cultures. Furthermore, he observed that the tubercle bacilli were present in abundance in the droppings of the flies and therefore it is obvious that the fly can act as a vehicle for tubercle by depositing its infected droppings on articles of food. Although the fly is only a simple vehicle and does not itself become tuberculous, its digestive fluids appear to be a favourable medium for the cultivation of the tubercle bacilli, and if so, why not for other pathogenic organisms? These experiments lend added importance to the necessity for the disinfection and destruction of tuberculous sputa. In connection with tuberculous sputa, mention may be made of a paper by Ziesche,[2] whose observations on the disinfection of droplets by coughing consumptives are of interest. He found that the droplets originating in the oral mucosa were free from tubercle bacilli, while in the drops originating from the bronchial mucosa bacilli were usually found, often in great quantities. The droplets were collected on a glass screen 18 c.c. square, and 40 c.c. to 80 c.c. distant from the patient. In 80 per cent. of the cases no bacilli, or less than 400 bacilli, were present; in the remaining 20 per cent. between 400 and 20,000 tubercle bacilli were found. He concludes by saying that drop infection would not occur if one remained in the presence of a consumptive person only for a short time, but a constant and close intercourse, as between mother and child, often leads to infection.

Reference may be made to the channels of infection of tuberculosis. For man the opportunities for infection vary according to customs and habits. Under certain circumstances children may be infected through the intestinal tract when consuming tuberculous milk or butter, or by putting their fingers tainted with tuberculous sputa into their mouths.

This theory of the intestinal channel of infection first received support from Behring. A very extensive source of infection is represented by the frequently large quantities of droplets containing tuberculous bacilli which are coughed out by the tuberculous patient and which mix with the atmosphere of his immediate surroundings. This is the most dangerous mode of transmission, as infection occurs even from the smallest quantity of bacilli, and, as the opportunities for this method of infection are so common, it is certain that by far the largest number of cases of human tuberculosis are the result of the inhalation of the tubercle bacilli ejected in the sputum droplets of tuberculous patients.

Dr. Ribbert, of Bonn, in a paper read at the International Congress of Hygiene in Berlin, states that intestinal infection does not play any great part as compared with aerogenic infection, and that tuberculosis of the bronchial glands can only be of aerogenic origin. Ravenel of Philadelphia, on the other hand, says that the alimentary tract is a frequent portal of entry for the tubercle bacillus.

In this connection Calmette's[3] conclusions require notice:—

1. There is considerable difficulty in experimentally showing the respiratory method of tuberculous infection.

2. In natural contagion dry dust containing the tubercle bacillus does not play any rôle in infection.

3. Ingestion of virulent tuberculous material or cultures in fine liquid emulsion constantly succeeds in producing tuberculosis in all susceptible animals. The bacilli can be absorbed by the intestinal mucous membrane without producing any lesion. They are carried by the chyle to the mesenterie lymph glands. Thence they are carried by the macrophages along the thoracic duct to the blood circulatory system. The capillaries of the lung are most exposed to infection, and this explains the frequency of pulmonary infection.

4. The course of tuberculous infection is the more rapid and grave according to the number of virulent bacilli absorbed, and greater when the absorptions occur at frequent intervals.

5. A closed tuberculous lesion resulting from a single infection is capable of cure, with a resulting immunity.

6. Inherited tuberculosis is rare, results always from infection *in utero*, and is of little importance as a factor in the origin of tuberculosis.

7. The notion of tuberculous soil or predisposition should be abandoned, as it has been shown by experiment that infection is always possible in susceptible animals, and it bears a direct relation to the number of virulent bacilli absorbed and to the frequency of the inoculation.

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[1] André, L. (November, 1906). *C.R. Soc. Med. Hop. de Lyon.*

[2] Ziesche, H. (1907). *Zeit. für Hygiene*, Vol. 57, No. 1, p. 50.

[3] Calmette, A. (September, 1907), "Les voies normales de pénétration du virus tuberculeux dans l'organisme." *Bull. de l'Institut Pasteur.*

* Article not consulted in the original.

uberculosis *—continued*

Nield Cook[1] called attention to the large amount of tuberculosis in Calcutta; and as bovine tuberculosis in India is rare, the channels of infection from milk and meat can practically be excluded. Schroeder, in an interesting paper, states that 40 per cent. of dairy cows that retain the appearance of health and are not known to be affected till they are tested with tuberculin, actively expel tubercle bacilli from their bodies in a way dangerous to the health of other animals and persons, and that tuberculous cows do not expel tubercle bacilli till some time after they contract the affection. The practical importance of this is that herds of tuberculous cattle can be cleaned by the periodic application of the tuberculin test.

Schroeder[2] draws attention to the fact that a considerable proportion of the dairy products are infected with tubercle bacilli owing to the frequency with which cow fæces are found in milk, for it has been proved that the commonest way for tubercle bacilli to pass from the bodies of tuberculous cows is with their fæces, and once milk is contaminated with tubercle bacilli, the latter enter the various articles of diet prepared from it, and are specially numerous in butter, in which they may remain alive seven weeks or longer without diminishing in virulence. He points out the usefulness of the tuberculin test, and the desirability of separating all reacting animals. Some interesting experiments were conducted by Oberwarth and Rabinowitsch[3*] to disprove the assertion that the appearance of tubercle bacilli in the lungs and bronchial lymphatic glands after introduction into the alimentary canal was due to aspiration.

Experiments were carried out in young guinea pigs, tubercle bacilli being introduced by laparotomy into the stomach. Some hours later tubercle bacilli were found in the lungs; but the objection was raised that regurgitation and subsequent aspiration might have taken place, so further experiments were carried out, gastrostomy being performed, and four weeks after, the œsophagus was cut at the neck and connected with the skin so that there were now two fistulous openings separated from each other by a bridge of skin.

The animals then received a feed of dried tubercle bacilli, the œsophageal fistula being closed by cotton wool and collodion to prevent regurgitation. In every animal tubercle bacilli were found in the blood, lungs and other organs, and in one animal 22 hours after the experiment. The result of these researches proves that virulent tubercle bacilli may be absorbed by the mucous membrane of the alimentary canal into the blood and may be deposited and possibly become latent in the lungs and other organs. Whitla,[4] in a paper entitled "The Etiology of Pulmonary Tuberculosis," discusses all the recent work on human and bovine tuberculosis, and has confirmed the results obtained by Calmette. Amongst other points, in an interesting paper, he notes that it has been definitely proved that the tubercle bacillus can pass through the intestinal mucosa like the fine particles of China ink without causing any lesion or leaving any local evidence of its point of entrance. McCaw, in an interesting paper, suggests that the following rules should be formulated in order to protect the State from the ravages of tuberculosis.

(1) Compulsory notification of births within 24 hours.

(2) Complete control of the milk supply by the State, ensuring the removal of dairies from the centres of large towns and cities; cleanliness in the collection of milk and in the transmission of it from the dairy to the consumer; the application of the tuberculin test to dairy cattle and the removal of such as react to this test.

(3) Medical inspection of school children and school premises to ensure sufficient hygienic measures and sanitary arrangements.

(4) Housing reform; thus raising the social and domestic conditions of the poor.

(5) Segregation of advanced cases.

(6) Compulsory notification of the disease.

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[1] Nield Cook, J. (November, 1907), "Tuberculosis in Calcutta." *Journal of Public Health.*

[2] Schroeder, E. C. (March 31st, 1908), "The Unsuspected but Dangerously Tuberculous Cow." *Journal of Comparative Pathology and Therapeutics.*

[3] Oberwarth, E., and Rabinowitsch, L. (1908), "Infection with Tubercle Bacilli which have been absorbed from the Alimentary Canal." *Berlin Klin. Woch.*, No. 6, p. 298.

[4] Whitla, W. (July 18th, 1908), "The Etiology of Pulmonary Tuberculosis." *Lancet*, Vol. II.

* Article not consulted in the original.

Tuberculosis —*continued*

A very interesting and instructive paper by Roberts and Bhandarkar on the existence of an acute tuberculous fever in India, leads one to think that this pathologial condition in the Tropics requires investigation (*see* "Fevers," *page* 66). As regards tuberculosis in the Sudan, where the disease is far from uncommon, Cummin's paper in the *British Journal of Tuberculosis* for January, 1908, may be consulted. Bovine tuberculosis is rare or unknown.

Typhus Fever. In the days of Baker and Schweinfurth typhus is said to have been common in Khartoum. In 1864 and in previous years a malignant form of the disease was reported from the old city. The Mahdi is said to have succumbed to it at Omdurman, but there is reason to suppose that he really fell a victim to cerebro-spinal meningitis. Indeed, it is quite possible that in those days typhus was confused with this disease and with relapsing fever, but, at the same time, available records show that the conditions favouring typhus were by no means lacking. These, according to Sandwith,[1] are (1) Overcrowding, (2) Deficient ventilation, (3) Uncleanliness, (4) Faulty conservancy arrangements, (5) Insufficient diet for the work required.

He thinks that it is likely to occur in the Sudan in the spring months, but from what one has seen of typhus elsewhere one would be inclined to expect its appearance during the winter months, which are sometimes comparatively cold. During this season the natives are apt to huddle together for warmth, and, contrary to their usual habits, to sleep indoors, so that certainly overcrowding and deficient ventilation are present.

I understand, however, that since this country was reconquered typhus has not been reported, and in some measure this is possibly due to the absence of famine conditions and of vagrancy.

Sandwith describes the disease as encountered in Egypt. He thinks that in the Sudanese hypostatic congestion is to be regarded rather as a symptom than a complication, and one which may materially aid the diagnosis. The adynamic is the most common form, the nervous type with marked cerebral symptoms being met with in better class patients. It appears that it may occur along with relapsing fever.

Sandwith compares the characteristic odour to that emanating from a cupboard of well-blacked boots, and I can confirm the aptness of this analogy, for there is a peculiar "acid" flavour or "tang" about it which is reproduced by the "boot" smell and by nothing else with which I am familiar.

The cause is still unknown, although recently in Egypt a protozöon, *Babesia hominis*, was described in 1903 by Gotschlich[2] as being the etiological factor. This requires confirmation.

Husband and MacWatters[3] have a paper on the disease as encountered in Northern India. They think it is more common than is generally supposed, having sometimes been mistaken for epidemic pneumonia. The diagnosis from pneumonia is difficult, and they came to rely chiefly on the following points:—

1. Characteristic mental condition—the patients being usually apathetic, dull, stupid and drowsy. 2. The dry, swollen and cracked tongue, caked with a patchy, rather thick, brown or even black deposit on the dorsum. The sides and tip were often comparatively free, but red and sore-looking. Occasionally, however, the tongue remains healthy. 3. The rash; but it is sometimes entirely absent, and even when present is indefinite, evanescent and difficult to see on a dark skin. 4. The blood examination. The differential leucocyte count differing from that of most other fevers; leucocytosis being present, and an increase in the red corpuscles.

Details are promised later. The authors favour the protozoal origin of the disease, and cite Sambon's belief that typhus and Rocky Mountain fever, in the latter of which a piroplasm has been demonstrated, are identical. At the same time, they were unable to find any blood parasite in their cases. They are inclined to believe that bed-bugs convey the virus, and this view is upheld by Hepper's[4] observations in Peshawar, which showed that out of six cases five had been exposed to the attacks of these insects, and that the outbreak ceased when all the bugs were killed and infection by them was rendered impossible. He admits, however, that there is as yet no definite proof.

Horiuchi[5] mentions this discovery in a recent paper on an outbreak of exanthematic

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[1] Sandwith, F. M. (London, 1905), "The Medical Diseases of Egypt." Pt. I.

[2] Gotschlich, E. (1903). *Deut. Med. Woch.*, Bd. XXIX., 329-331.

[3] Husband, J., and MacWatters, R. C. (June, 1908), "Typhus Fever in Northern India." *Indian Medical Gazette.*

[4] Hepper, E. C. (June, 1908), "An Outbreak of Typhus Fever in Peshawar." *Ibid.*

[5] Horiuchi, T. (May 16th, 1908), "Ueber einen neuen Bazillus als Erreger eines exanthematischen Fiebers in der Mandschurei, etc." *Cent. für Bakt. Origin.*, Abt. 1, No. 7, Bd. XLVI.

Typhus Fever— *continued*

fever, evidently closely allied to typhus, amongst the Japanese troops in the Russo-Japanese war. From the stools, and in some cases from the urine, he isolated an organism, the *Bacillus febris exanthematicus mandschurici*, which he regards as the cause of the disease, and which resembles organisms of the paratyphoid group, and whose cultural characteristics he describes in detail.

Rogers[1] gives statistics to show that true typhus still occurs in India, but it is now rarely seen, owing to the improved sanitation of gaols in that country.

Newsholme[2] discusses at length the relation of poverty to typhus in Ireland. He says of the operative causes of typhus, specific infection is admitted generally to be indispensable. Malnutrition has not always been associated with epidemics of this disease, and there has been no constant association of epidemics with exceptional overcrowding, or cessation of epidemics when overcrowding has been enormously reduced. Vagrancy is the one factor which has always accompanied specific infection, and in the absence of which epidemics have failed to occur even in the vicinity of infected populations.

He also points out that the suppression of typhus in Ireland had been due chiefly to the efficient immobilisation of infection by means not intended expressly for that purpose.

M'Vail[3] has an interesting account of typhus, chiefly as regards its occurrence in Glasgow. He mentions its association with fleas, which have been suggested by Matthew Hay as carriers of the infection. He gives a plan of a useful type of reception-house.

Vaccination. In the Tropics small-pox spreads with a rapidity and attains a severity which is rarely seen at home, and, as most tropical countries are beyond the pale of the "Conscientious Objector," one has opportunities of noticing the good results from vaccination, while in some parts even the mind of the ignorant and prejudiced native is impressed by its efficacy when an epidemic presents itself in his village.

Fink[4] supplies some striking and interesting evidence in favour of vaccination in Burmah. He noted that it was a common experience where small-pox is epidemic to find the local medicine man inoculating all children, who have not been protected by a previous attack of the disease. The method consisted in selecting a mild case, removing the scabs off the pustules, grinding these scabs down to a fine powder, mixing with water, and injecting some of this mixture into the forearm or rubbing it into open abrasions.

In a village in the Pakokku district in Burmah, where small-pox had broken out, 59 persons had been attacked: 22 of these, mainly children, had got the disease by infection, and the rest, viz., 37, by inoculation. Four deaths occurred among the children who had not been inoculated.

After personal experience of each child vaccinated in 1900 and 1901, and also of all those inoculated, Fink observed that not a single child successfully vaccinated a year or two previously got small-pox, either by infection or by inoculation. His figures are worth quoting:—

Number of children successfully vaccinated in 1900 and 1901—144.

Number successfully vaccinated, inoculated without result—123.

Number successfully vaccinated and have resisted infection, but were not inoculated—21.

Nield Cook,[5] in an excellent paper, describes the method employed by him in the cultivation and preservation of calf lymph in Calcutta.

Climatic difficulties occur in the Tropics in connection with the cultivation and preservation of calf lymph.

Blaxall and Fremlin's[6] observations go to show that a vaccine is rendered inert by exposure to a temperature of 37° C. for twenty-four hours, but it will stand a temperature of 180° C. for several weeks without deterioration, and can be kept for a year or more in cold storage at a temperature of a few degrees below zero centigrade without any loss of

[1] Rogers, L. (London, 1908), "Fevers in the Tropics."

[2] Newsholme, A. (November, 1907), "Poverty and Disease, as illustrated by the course of Typhus Fever and Phthisis in Ireland." *Proc. Roy. Soc. Med. Epid. Sect.*, Vol. I., No. 1.

[3] M'Vail, J. C. (London, 1907). "The Prevention of Infectious Diseases."

[4] Fink, L. (July 16th, 1904), "The Efficacy of Vaccination tested by Inoculation and Small-Pox." *British Medical Journal.*

[5] Nield Cook, J. (May, 1907), "The Cultivation and Preservation of Calf Lymph." *Indian Medical Gazette.*

[6] Blaxall, F., and Fremlin, H. (August 9th, 1906), "Glycerinated Calf Lymph." *Lancet*, p. 669.

potency, although, if kept at the approximate temperature of an ice-box, viz., 10° C., it loses its activity as a vaccine to a certain extent. **Vaccination** *—continued*

Nield Cook's method, employed in Calcutta, consists in making enough glycerinated lymph to last till the next cold weather, the vaccine being poured into test-tubes nearly up to their rim. These are sealed by sterile corks which are pushed in so as to squeeze out a little vaccine, and thoroughly waxed over. The test-tubes are then placed in tin cases, which are sealed up and put away in a tin box, and this is stored in an ice company's cold storage at a temperature of 5° C. Nield Cook considers that rabbits are valuable additions to a lymph depot. He makes use of the rabbit:—

(1) To estimate the strength of the vaccine employed by inoculating the shaved backs of rabbits with 1 c.c. of varying dilutions of vaccine. If the crop of vaccine is of excellent quality the eruption produced by a dilution of 1 in 500 is still confluent.

(2) To renovate the vaccine. If a stock of vaccine be rapidly passed through a series of calves, it soon begins to deteriorate—especially under unfavourable climatic conditions.

Pulp may be taken from a rabbit on the fourth day, diluted with glycerin, and this glycerinated rabbit vaccine used for the vaccination of calves.

(3) The rabbit may be used to observe whether the vaccine is acting efficiently.

Vaccine in India is preserved either in lanoline, glycerin or chloroform. Lanolated vaccine has the disadvantage that micro-organisms may grow in it after it is made, although claims are made for it that it retains its potency under unfavourable climatic conditions. Vaccine preserved in chloroform resists the ingress of bacterial impurities, and, owing to the rapid bactericidal action of chloroform, this method of preservation is useful if vaccine is required in a hurry, as it takes some time for the germicidal action of glycerin to develop.

Hans Ziemann[1] has a very practical article in the *Berliner Klin. Wochenschrift*, 1908, No. 3, on "Protective Inoculation against Small-pox in the Colonies." This observer advises that only such vaccines should be employed for vaccination as are subject to Government control, and that an institute for the preparation of lymph should be established in every colony. Care should be taken to select healthy young calves, three to six months old. It is advisable to vaccinate a human subject with lymph freshly imported from Europe, and then to use this humanised vaccine with which to inoculate the calf. In localities where no calves are available, efficacious vaccine, similar to calf vaccine, may be obtained from buffaloes, gazelles, camels, dogs, horses, donkeys, pigs, monkeys, guinea pigs and rabbits.

Ziemann further recommends that the technique of vaccination carried out should be the same as in Europe, and should be performed in the cool season or during the coolest hours of the day. He holds that vaccination is successful if one well-developed pustule results.

Native adults should have six incisions on the left upper arm, while eight on those of children will suffice. If sufficient calf lymph be not available, arm-to-arm vaccination may be performed as long as no doubts exist as to the liability of infection through relapsing fever or sleeping sickness. The vaccine should only be taken from healthy children up to about eight years of age. Ziemann strongly recommends an important practical point, viz., that every colony should have travelling doctors appointed to undertake the systematic vaccination of the population, especially on caravan routes, and that native assistants should be instructed in the technique of vaccination. As regards the Sudan, the benefits conferred by vaccination are much appreciated by the natives, who in the past have suffered severely from the ravages of small-pox, and are only too willing to submit to such an efficient preventive measure. At present all vaccine lymph is imported into the Sudan, stored on ice and issued as required. It appears to be of a satisfactory quality, but doubtless it would be better to have a vaccine institute established in the country, especially for the supply of lymph to the more distant provinces.

Veterinary Diseases. The subject of veterinary diseases is one of very great economic importance in the Tropics. Some of the diseases affecting animals have already been considered, and, as regards these, there is no necessity to reiterate.

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[1] Ziemann, H. (August 15th, 1906), "Small-pox in the Tropics." *Journal of Tropical Medicine and Hygiene*, Vol. XI., No. 10, p. 159.

eterinary iseases— *continued*

Bilharziosis in Animals.—In a long paper by Montgomery[1] there is an account of bilharziosis of the horse, which he says is widely distributed throughout the Himalayan districts of India. It is of interest to note that in no cases were the ova of the *Schistosomum indicum* found in the centrifugalised urine of ponies kept under observation, although the adults were found on post mortem examination of the fæces and scrapings of the rectal mucous membrane. Ante mortem examination was also fruitless. The post mortem appearances were marked congestion of the portal venous system, the mesenteric veins of the colon and rectum being distended and varicose; careful dissection of these revealed the parasite. The pelvic veins were very congested and tortuous. The bladder showed varicosity of the veins of the neck, and punctiform hæmorrhages in the fundus of bladder. The mucous membrane was intact and free from any papillomatous growth.

The large intestine of equines was the only organ in which ova could be found present in any numbers. They lay between the Lieberkuhn glands. The mucus showed petechial hæmorrhages. Scrapings of these hæmorrhagic areas revealed the presence of eggs.

The *Schistosomum* is best obtained by dividing the veins of the portal system and collecting the blood in a tray. The male parasites may be detected as small white bodies which, if the autopsy has been performed soon after death, may be seen undergoing various changes in shape. Montgomery obtained the *Schistosomum* twice in the pancreatic veins and once in the pelvic veins. Of interest is his observation that the ova found in the rectum had terminal spines despite the powerful muscular wall of the horse's rectum. Further, Montgomery describes the presence of the *Schistosomum indicum* in the portal vein and its branches, and in the pancreatic vein of a donkey that died from surra. The lesions presented post mortem were analogous to those found in the horse. The *Schistosoma* in the donkey were larger and longer than those found in the horse.

Montgomery[1] made some further investigations on bilharziosis in cattle and sheep in India. The observations of Sonsino and Bomford show that in cattle serious and extensive lesions may occur as a result of bilharziosis, viz., intestinal catarrh, thickening, ecchymoses and œdema in the region of the ileo-cæcal valve. The mucous membrane of the bladder was ecchymosed, and contained papilliform elevations. In all the lesions the characteristic ova of *Schistosomidæ* were present.

Montgomery did not find these lesions in any of the Muktesar cattle, excepting in one case which displayed an interesting pathological condition of its lower bowel, two varieties of hæmorrhagic lesions being present. In the variety composed of minute discrete points, bilharzia ova of the human type were found. In the other variety of hæmorrhagic lesions, which were linear and were arranged transversely to the longitudinal fold of mucous membrane upon which they were situated, spindle-shaped eggs of the *S. bovis* type were discovered.

Two new species, the *S. bomfordi* and the *S. spindalis*, were found in the mesenteric vessels of a bull and of two plains cattle, respectively.

Sonsino discerned the *S. bovis* in sheep in Egypt, in 1876, and since then the parasite has been found in India. Montgomery observed no gross pathological lesions in the sheep, and no ova were discovered, and yet in one of his cases found practically every vessel in the portal system occupied by adult bilharzia parasites.

Baldrey ascertained the presence of bilharzia ova in a sheep which died at Lahore. The eggs were of the human type, resembling *S. indicum*. In a second sheep the same observer found a further variety of ova differing from the others in the shape of the spine, but no adults were discovered.

Reference may be made to *Bursati*, a strange complaint associated with the presence of filaria embryos in the skin and connective tissues in horses, which is common in India.

Lingard[2] concluded from a number of observations made in the Muktesar Laboratory on horses and cattle, that the filarial embryos are present in the blood of affected animals in varying numbers during the twenty-four hours, and that between six o'clock and ten o'clock in the evening the numbers of these embryos increase enormously. They were fewer in number in the month of September as compared with the months of June and July. These embryo filaria are chiefly found in country-bred horses, but also occur in Arabs and Walers. The mature filaria have their habitat in the walls of the larger arteries. It is

[1] Montgomery, R. E. (April, 1906). "Observations on Bilharziosis among Animals in India." *Journal of Tropical Veterinary Science*, Vol. I., p. 138.

[2] Lingard, A. (October 1st, 1905), "The Bursati of Horses, a Filarial Disease." *Indian Medical Gazette.*

possible for these filaria to circulate in the peripheral blood in large numbers without causing any cutaneous lesions or producing untoward symptoms.

These embryos may disappear entirely from the blood and may become lodged in the internal organs. The *Bursati* sore, or ulcer, contains characteristic yellow-coloured granules, the so-called *Kunkar*. These kunkar nodules have been found to be made up of large numbers of filarial embryos clumped together, and it is believed by Dr. Lingard that these parasites cause *Bursati*.

Glanders.—Although this disease is fairly prevalent in tropical countries, it is beyond the scope of this work to consider the whole subject of glanders. It has occurred in Khartoum, and *B. mallei* has been isolated.

Reference may be made, however, to mallein as a diagnostic aid in glanders.

Discovered in 1890 by a Russian veterinary surgeon, it has furnished an almost infallible means of diagnosing the existence of the disease in its latent stages. When injected into a healthy animal no symptoms appear, but when glanders is present there is not only a characteristic febrile disturbance, but a swelling at the site of the injection. The intensity of the reaction, however, bears no relation to the extent or duration of the disease.

Accordingly it has always been held by the veterinary profession that the mallein reaction is absolutely specific. Schattenfroh, however, in 1894, showed, as the result of some tests on guinea pigs, that mallein acted similarly to various bacterial proteins; but the mallein used by Schattenfroh consisted of protein precipitated by alcohol from concentrated broth cultures of *B. mallei*, so that his conclusions do not apply to mallein as it is now prepared.

Südmersen,[1] in an interesting paper, describes his observations on some non-specific reactions of mallein. This observer noted that in one experiment on a healthy horse that had undergone treatment with diphtheria toxin for over two years, a large local reaction and a rise of temperature, sufficient to condemn the horse, was obtained after an injection of 0·25 c.c. of mallein. A second dose of 2 c.c. mallein was injected within three days of the first injection, the local swelling was more marked than before, and the temperature reaction was similar. The reactions obtained were considered sufficient to condemn the horse as glandered, but a post mortem revealed no evidence of glanders. The deductions from Südmersen's observations are that horses immunised against other bacteria or bacterial products will give a large local reaction to mallein, but this is usually not associated with a rise in temperature. This local reaction disappears rapidly, and can thus be distinguished from the reaction in the case of glandered horses. In the few cases when a rise in temperature took place the curve was markedly different from that obtained in the case of glandered animals. Other bacterial products react similarly to mallein upon immune animals. Vallee[2]* states that among glandered horses subjected to the skin reaction by means of mallein he observed phenomena similar to those produced by the action of tuberculin on tuberculous animals. The glanders skin reaction may give rise to erroneous interpretation; thus, some horses that were not glandered were able to produce in the region of the skin of the neck a distinct reaction. The glanders ophthalmo-reaction is not so distinctive as to induce one to prefer it to the subcutaneous inoculation of mallein.

An interesting case of a disease similating farcy in a horse, from which an organism possessing many features in common with the *Bacillus mallei* was isolated, is recorded by Baldrey and Martin[3] in India. The horse was tested with mallein, but there was no rise of temperature and the local reaction was slight. Smears made from pus revealed a few organisms resembling *B. mallei* in size and shape, but somewhat shorter and more rounded. The cultural and inoculation tests were not specific of glanders.

Reference may be made to *Rinderpest*, a disease which, owing to its ravages in certain parts of the Tropics, has caused considerable loss to the industrial community, and has been the means of crippling trade. This is especially the case in the Sudan,

[1] Südmersen, H. J., and Glenny, A. (January, 1908), "On some Non-specific Reactions of Mallein." *Journal of Hygiene.*

[2] Vallee, A. (June 30th, 1907), "Sur la cuti-réaction et Ophthalmo-Réaction dans la Morve." *Bull. Soc. Cent. Med. Vet.*

[3] Baldrey, F. P., and Martin, G. D. (July 1st, 1906), "A disease of the Horse similating Farcy, from which an Organism possessing many features in common with Bacillus Mallei was isolated." *Journal of Tropical Veterinary Science*, No. 3, p. 316.

* Article not consulted in the original.

Veterinary Diseases— *continued*

where it is constantly being re-introduced from Abyssinia and Erythrea. The attempts to isolate the organism causing this pest have so far been unsuccessful, although it is certain that the infecting agent is contained in the excretions of the nasal passages, conjunctiva, intestines, and in the blood of infected animals, for with any one of these the disease can be reproduced in other individuals. The virus of rinderpest from the blood or any of the secretions possesses but little viability. Left at a fairly warm room temperature it becomes inactive; but it retains its activity longer when it is placed on ice. The length of time during which it may be kept in a viable condition varies according to reports between three and thirty-two days, but Woolley's[1] observations indicate a limit of from three to six days. At a temperature of 36° to 40° C. it becomes inactive after two days, according to Theiler. According to Koch, dried blood may remain virulent for four days. In the presence of other organisms it is killed, and sunlight destroys it in five minutes, according to Braddon, but ordinary daylight has little effect. Yersin observes that the filtrate obtained by passing rinderpest blood through Chamberland candles can cause the disease.

There is a difference of opinion regarding the characteristic lesions of this disease, no doubt due to the fact that the European, South African and Philippine types of rinderpest differ in many points. The most constant morbid changes in the body are emaciation, sero-purulent conjunctivitis, congestion and general inflammation or ulceration of the gastro-intestinal tract. Petechiæ present themselves in the heart, kidneys, liver and lymphatic glands. The classical cutaneous eruptions and ulcerations of the nasal and oral mucous membranes present in the European disease are rarely seen in the Philippine variety. As regards blood changes, Baldrey[2] found leucocytosis immediately after infection in uncomplicated cases of rinderpest.

There are but two methods of preventing the entrance and spread of rinderpest in a country. These methods are immunisation and quarantine.

The methods of producing immunity to rinderpest are three in number, namely, the active, passive, and a combination of the active and passive. The active immunity is larger than the passive, and in animals which have recovered from the natural disease it is apparently permanent. Of the methods of producing active immunity mention may be made of Koch's bile method. Ten cubic centimetres of bile taken from an animal on the sixth to the eighth day of the disease will, upon injection into a non-immune animal, produce an immunity which persists for several months. All investigations have not had as favourable results as those of Koch; those of Lingard and Rogers, for instance, were negative.

The advantages of this method are that no reactionary fever, suppression of lactation, or abortions occur, and the operations can be carried out in the field. The disadvantages are that immunity is not established till the tenth day after inoculation, nor is the immunity permanent, and it has no curative properties. Holstock and Edington modified Koch's method, but without any particular advantage. The method of producing passive immunity originated in the observation that the serum obtained from naturally salted cattle possessed a slight immunising power. Kolle and Turner found that by injecting increasing amounts of virulent blood into salted animals they were able to produce an efficient serum which could be used successfully in doses of 10 c.c. to 20 c.c. The advantages of this method are that it has no detrimental effect on pregnant or dairy cattle, that it will favourably modify a disease of one to three days' duration, and, further, the serum will keep well for a long time. Of the combined methods of immunisation, mention may be made of the serum-simultaneous method and the deferred virulent blood method.

In the serum-simultaneous method the animal is inoculated with 1 c.c. of virulent rinderpest blood under the skin on the one side and a minimal efficient dose of immune serum on the other. A mild attack of the disease follows and the animal recovers. About 90 per cent. of cases treated in this way display an immediate permanent immunity, while in the remaining 10 per cent. the immunity produced is only a transient one.

The chief disadvantage of this method is the danger of introducing latent blood diseases such as Texas fever, trypanosomiasis, etc., and, further, lactation and pregnancy are frequently interfered with, while there may be a difficulty in obtaining virulent blood.

In the deferred virulent blood method the serum is given a day or several days before the introduction of the virulent blood, the temperature being observed. If no reaction

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[1] Woolley, P. G. (June, 1906), "Rinderpest." *Journal of Tropical Veterinary Science.*

[2] Baldrey, F. S. (June, 1906), "Some Observations on Normal Rinderpest Blood." *Journal of Tropical Veterinary Science*, p. 47.

follows, an inoculation of 10 c.c. virulent blood, and a further inoculation of another 10 c.c. is given. The advantage of this method is that the mortality is less and more reactions are obtained than by any other.

It is necessary to standardise the serum, and that amount should be given which will allow a febrile reaction to take place, but after the administration of which the animal shows no other symptoms of the disease.

In addition to the simultaneous method and its modification, Danysz and Bordet used defibrinated virulent blood, but the disadvantage of this is that the material must be fresh and free from other pathological micro-organisms.

Littlewood,[1] from his observations in Egypt, says it is possible that serum when injected into imported animals does not afford the same protection against rinderpest as it does in native herds, especially if these animals have been exposed to adverse conditions.

Woolley believes that by a judicious use of serum, and simultaneous methods, epidemics may be controlled, and the cattle in a district immunised. In the absence of serum, glycerinated bile or defebrinated immune blood may be used. All infected animals should receive intravenous injection of serum, but in order that it may have every chance of success it must be applied before the third day of the disease. Not less than 100 c.c. should be injected, and it is possible that several injections may be necessary. In India the serum method alone is almost exclusively used. It was found that in using the simultaneous method the small hill breeds of cattle in India require 15 to 18 times the quantity of anti-rinderpest serum given to the ordinary breeds of plains cattle. Half-bred hill and plains hybrids require to be treated with amounts varying from 15 to 18 times the quantity required for plains cattle, and Lingard pointed out that a grave responsibility was incurred in protecting cattle of different breeds without some knowledge of their respective susceptibility. This factor, amongst others, prevented the more extensive adoption of the simultaneous method in India, and the large number of inoculations are now performed in actual outbreaks with serum alone, while, to ensure success, Walker[2] suggested a ready method of estimating the proper dose of serum in the field. The serum made at the Imperial Bacteriological Laboratory is tested on hill cattle, and it is prescribed at so much for hill cattle up to 600 lbs. in weight and $\frac{1}{15}$th of the dose for plains cattle up to 600 lbs. in weight. Walker's method briefly consists of estimating the mortality per cent. before inoculation. If the mortality before inoculation is less than fifty per cent. the standard dose for plains animals is to be given. If the mortality is more than fifty per cent. and less than 75 per cent., double the dose for plains animals is to be given. If the mortality is over 75 per cent. and under 85 per cent. five times the dose is required. If the mortality exceeds 85 per cent. the full dose for hill animals, *i.e.* eighteen times that for plains animals is to be given. Although a somewhat rough method of estimating the dose required, it has been practised with success in the Punjab. All contact animals in an outbreak are inoculated with a sufficiently large dose of the serum to protect them from an attack of the disease, healthy and infected animals are mixed together, and disinfection of the stables and steadings is carried out. In the Sudan the serum method alone is now employed and with great success.

Horse Sickness. This disease has caused a considerable mortality amongst horses and mules in South Africa.

It is an inoculable disease of horses and mules, but is not contagious. In the Transvaal it is a disease of yearly incidence, occurring for the most part after the beginning of the rainy season in summer, especially in low-lying stretches of country formed by the bush veldt. There appears to be a certain connection between the relative altitude of a locality and horse-sickness, for in the highest parts the disease is least known. It disappears quite suddenly at the beginning of the cold season.

The micro-organism of horse-sickness has not yet been discovered, and the method of transmission of the virus has yet to be elucidated.

The opinion that the dew is concerned with the production of the disease is not held by Theiler,[3] who is inclined to lay some stress on the part played by blood-sucking

[1] Littlewood (1905). *Journal of Comparative Pathology and Therapeutics*, p. 312, Vol. XVIII.

[2] Walker (February, 1908), "A Practical Method of determining the dose of Serum required to protect Contact Animals in outbreaks of Rinderpest." *Journal of Tropical Veterinary Science.*

[3] Theiler, A. (May, 1903), "Some Diseases of the Horse in South Africa." Bulletin 2.

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insects, especially mosquitoes. This view certainly receives some support, from the following facts associated with the disease. Horse-sickness is mostly contracted after sunset, when insects begin to fly and become troublesome, the disease may travel from the low veldt to the high veldt in certain rainy seasons which are favourable for the breeding of insects; further, stable-kept horses are less likely to be infected than others; and, lastly, the disease tends to disappear suddenly, as a rule, after the first frosts.

Theiler[1] elucidated an interesting fact, namely, that horse-sickness can be transmitted to dogs and that the virus can be transmitted from dog to dog. The post mortem lesions found in dogs are identical with those in the horse. Some difficulty was experienced in explaining the fact that horse-sickness may be contracted in parts of the veldt where an equine had not been for some years, but in view of the fact that other animals besides equines can suffer, this difficulty has now been overcome.

Theiler and Simpson undertook a series of experiments with mosquitoes, *Culex*, *Stegomyia* and *Anopheles*, in order to see whether they would act as hosts for the horse-sickness virus. The experiments, unfortunately, were not a success, as the stable atmosphere was inimical to the well-being of the mosquito. This observation is of interest, as it is quite in accordance with the theory of the transmission of horse-sickness by mosquitoes. Stable horses do not as a rule contract the disease.

It still, however, remains to be proved whether the mosquito is the host of horse-sickness, and, if so, to discover the particular species.

The question of the identity of horse-sickness with heart-water, a view brought forward by Edington, was disproved by Theiler in a series of experiments on goats and horses. Theiler conducted some further experiments with the immunisation of mules against horse-sickness, and found that the previous method of injecting the virus into the jugular vein can be abolished with safety and replaced by a subcutaneous injection, and that mules whilst undergoing horse-sickness reaction need not necessarily be stabled. There was no difference in the character of the reaction between stabled and non-stabled mules.

Watkins Pitchford,[2] in an interesting monograph on observations on the morbid anatomy of South African horse-sickness, concludes that the cause of death in horse-sickness is due to capillary embolism or its results, the emboli consisting of certain cells and masses of semi-crystalline pigmented material in great abundance.

He found that the medullary cavity of the thigh bones, and also sometimes of the arm bones, contain these cells and pigmented material in great abundance. The overgrowth of red marrow is probably the primary lesion of the disease. Further, he considers that the disease horse-sickness belongs to an entirely new class, to which some such name as critical myelæmic or myelocytic embolism might appropriately be given.

Horse-sickness occurs in the Sudan and has caused loss amongst cavalry horses at Shendi. I recall an epidemic in Kordofan.

Epizootic Lymphangitis is a virulent inoculable disease which is characterised by suppuration of the superficial lymphatic vessels, and is due to a specific organism. Owing to the scanty amount of literature relating to this disease, the references to it have been chiefly taken from a monograph on the subject by Pallin.[3]

This disease occurs in horses, mules and cattle, and is caused by a *cryptococcus* which is present in the morbid tissues and products, partly free in the plasma and partly enclosed in pus corpuscles, phagocytosis being marked. It is slightly ovoid, with one extremity pointed and the other rounded. It measures about 3–4 μ in diameter, and is characterised by having a clearly-defined contour with a refractile double outline. Tokishige, in Japan, places it in the class of Saccharomyces.

Theiler[4] describes it as a *Saccharomyces farciminosus*, belonging to the group of yeast fungi. There is often a small strongly refringent nucleus seen in the transparent cell body, which is frequently active, moving from pole to pole of the yeast cell. It is stained with difficulty, but can be coloured by the Gram-Weigert-Kuhne method or by the modification of Gram's method as used by Claudius. Ziehl's Solution of carbol fuchsine, composed of

[1] Theiler, A. (1905-6), Report of Government Veterinary Bacteriologist, Transvaal Dept. of Agriculture.

[2] Watkins Pitchford, E. (1904), "Observations on the Morbid Anatomy of South African Horse-Sickness."

[3] Pallin, W.A., "Epizootic Lymphangitis."

[4] Theiler, A., "Epizootic Lymphangitis." Transvaal Department of Agriculture, Bulletin No. 4, 1906.

fuchsine, 1 gramme; absolute alcohol, 10 c.c. and 10 c.c. of 5 per cent. aqueous solution of carbolic acid gives good results. The *cryptococcus* grows with difficulty in culture, and the growth is always slow and develops better in acid than alkaline media. Microscopically the colonies consist of conglomerated masses of hyphæ.

The incubation period of the disease is three weeks to three months, and the lesions may occur on any part of the body, but are most frequently associated with those parts exposed to wounds, kicks, contusions and harness galls. It commences as a small pustule occurring at the seat of a pre-existing wound with knotting of the adjacent lymphatic vessels, and later a long chain of nodules may be seen extending along the course of the lymphatics. This is followed by pustules and abscess formation. The nasal mucous membrane may become affected by papules or pimples developing into vesicles, and resulting in characteristic ulcers with a raised edge and dug-out centre. The condition has to be diagnosed from glanders, suppurative lymphangitis, botryomycosis, tubercle, lymphangitis and bursitis. The mallein test is useful, and if a reaction occurs the conclusion is that epizootic lymphangitis is complicated by the presence of glanders—probably chronic glanders of the lung. Glanders and epizootic lymphangitis can occur together in the same animal. The treatment involves itself into local, general and prophylactic, and consists in dissecting out the affected lymphatic vessels, efficient cauterising of the wound, and the administration of corrosive sublimate, half to one grain in solution. Prophylactic measures must be carried out in an efficient manner, all infected cases isolated or destroyed and cremated, suspicious cases isolated and kept under observation. Harness and saddlery used on infected cases should be thoroughly disinfected with perchloride of mercury solution, 1 in 250, or else should be burnt together with the grooming kit. Stables should be thoroughly disinfected. A shed in which an outbreak has occurred should be considered infected until at least six months have elapsed from the date on which the last case was discharged; further, the horses of the stud should be kept under observation for another six months.

Under the name "Sarraja," epizootic lymphangitis is commonly met with in the Sudan, especially amongst mules and horses. It has caused such serious loss that the Veterinary Department have issued a special pamphlet regarding it. The disease appears to occur all over the country. I have seen it both in the Northern and Southern Sudan. Captain Olver thinks that it was introduced by animals from Abyssinia and informs me (A. B.) that it is not so virulent as it was in England. He finds that in most cases early removal of the affected tissues is an efficient mode of treatment. The most recent paper on the subject is one by Pricolo, a translation of which will be found in the *Journal of Tropical Veterinary Science* for May, 1908.

Pricolo[1] has recently made a useful contribution to the literature on this subject.

Strangles. Reference may be made to the etiology of this disease, which has been worked at by Baruchello. His conclusions are that in complicated and simple forms of strangles a staphylococcus associated with a streptococcus is found.

Baruchello[2]* constantly found it in the morbid products or in the circulating blood in very severe clinical cases, in septicæmic forms of strangles with complications, and in the pleuro-pneumonia associated with strangles at all stages of the disease.

His method of isolating these organisms from the blood consists in shaving and careful disinfection of the skin over the jugular vein, which is transfixed by means of a pipette, and the blood is collected in a flask containing 10 per cent. citrate of soda. The mixture is then planted out in agar tubes.

Baruchello believes that the great variability in the clinical forms of strangles and in the gravity and complications which this disease exhibits, are generally the result of the combined action of the two microbes.

Strangles also occurs in the Sudan. The laboratory museum contains some excellent specimens of nodules in the lungs.

Botriomycosis. This disease is of special interest, as it occurs to a great extent in camels in the Sudan. A considerable number of these animals die from this cause in Kordofan.

Caused by an organism known as the *Botriomyces*, it affects chiefly horses, but has been known to affect cattle, swine, and even the human being. It usually results from a wound

[1] Pricolo, A. (May, 1908), "Contribution to the Study of Epizootic Lymphangitis." *Journal of Tropical Veterinary Science.*

[2] Baruchello, L. (April 15th, 1907). *Rev. Gen. de Med. Vet.*, No. 104, p. 433.

* Article not consulted in the original.

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infection, the lesion produced consisting of chronic inflammatory proliferation of connective tissue with suppurating foci; the whole constituting a botriomycoma. The pus from a botriomycoma is characteristic in its appearance, being of a sticky mucoid character and when spread on a slide a number of granules can be seen.

It differs from the pus found in actinomycosis, as the granules do not give a gritty sensation when rubbed between the fingers.

Microscopic examination shows that the granule is almost opaque and has an undulating margin. If the granule is still further broken up and stained by Gram, it is seen to consist of numbers of staphylococci, held together by some amorphous matrix. The absence of filaments and clubs distinguish this from actinomycosis.

The common sites for botriomycoma are the front of the shoulder, spermatic cord, elbow and udder.

As regards treatment, Woolridge[1] considers surgical measures should be resorted to as early as possible, and that the action of potassium iodide as a drug may be of use where the growths are so large or so diffuse as to be inoperable.

Sleeping Sickness in Fowls. A reference to this disease is of interest, owing to the presence of a micro-organism found by Damann and Manegold,[2]* viz., a *streptococcus* which is capsulated. This streptococcus grows well in ordinary media, especially on blood serum to which 6 per cent. glycerin has been added. The streptococcus retains Gram's stain and is pathogenic to pigeons, rabbits and other animals, and the above observers found that the disease may be communicated to lambs and dogs.

It was noticed in 1904 by the authors, the most striking symptom being marked somnolence, and on account of this they named it Fowl Sleeping Sickness.

The affected fowls appear dejected, with swollen eyes and pale combs and have symptoms of catarrh. They eat well and may move about after feeding, but as a rule they remain still and huddled together, sleeping for hours and days and leave off laying. Finally they become extremely emaciated and die.

The emaciation the authors attribute to the toxin of the *Streptococcus capsulatus gallinarum.* Besides symptoms of septicæmia, inflammatory changes occur in the intestinal canal. The incubation of this disease is eight to fourteen days, and the duration is about three weeks.

Influenza in Horses. Gray[3]* refers to this condition being caused by a *Cocco-bacillus,* which has been found experimentally to be fatal to the guinea pig, rabbit and horse when inoculated subcutaneously into these animals. This *cocco-bacillus* is decolorised by Gram's method of staining, is ærobic and never forms streptococci when grown in liquid media. The disease appears to be highly contagious and produces symptoms affecting specially either the pulmonary, cardiac, renal, or intestinal system or the central nervous system. Specific sera have been employed as prophylactic measures and for diminishing the severity of the symptoms.

Filariasis. Reference has already been made to the disease known as Bursati, possibly caused by filarial embryos.

Pease,[4]* in an interesting paper, describes a disease in horses simulating dourine, but found to be produced by filarial embryos. It is of interest to note that no adult filaria were discovered in his cases. The chief signs were the presence of œdematous patches resembling the plaques found in dourine, and exuding a pale straw-coloured serous fluid. In samples of blood removed from these patches, filarial embryos were present in abundance.

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[1] Woolridge, G. H. (September 30th, 1907), "Actinomycosis and Botriomycosis." *Journal of Comparative Pathology and Therapeutics,* Vol. XX.

[2] Damann, C., and Manegold, O. (1906). *Archiv für. Wissenschaft u. prakt. Tierheilk,* Bd. 33, S. 41-70; also (February, 1908), "Sleeping Sickness in Fowls." *Journal of Tropical Veterinary Science,* p. 128.

[3] Gray, H. (March 19th, 1904), "Influenza in the Horse." *Veterinary Record.*

[4] Pease, H. T. (October, 1906), "A Disease simulating Dourine caused by Filaria." *Journal of Tropical Veterinary Science.*

* Article not consulted in the original.

Mason[1] observed the presence of larval filaria in the blood of a camel in Egypt. Their presence in the peripheral blood was influenced to some extent by day and by night, as they were more easily found in smears taken late in the afternoon or at night. Veterinary Diseases—*continued*

Mason also noted the presence of worms in the testicles and blood-vessels and excretory tube of the epididymis of the camel.

These worms resemble the *Filaria papillosa*, but are larger. Mason concluded that these filaria in the testicles may be the mature forms of the microscopical larvæ found in the blood.

Water. Simpson,[2] in an interesting paper, draws attention to the following points, which may assist one in the search for water in an unknown locality. Water is more easily obtained in a hilly than in a flat country, and can usually be found under waterless rivers under the dry course of nullahs, at the junctions of ravines and valleys and at the foot of hills.

A well-wooded water-shed, and localities covered with vegetation and verdure, invariably favour the presence of water. In the absence of vegetation, particular localities over which fogs or swarming insects are noticed as a rule indicate water near the surface.

In limestone districts and at the foot of hills springs are generally to be found, and deep wells exist in localities abounding in sandstone. For drinking purposes, the ordinary sources of water supply may be divided into (1) rainfall, (2) water derived from shallow wells, (3) water derived from deep wells and springs, (4) surface waters, such as rivers, lakes and tanks.

Rainfall forms a very common source of supply in several parts of the Tropics.

The rain is, as a rule, collected in storage tanks, which receive the rain from the roof, or on large areas of ground which have been rendered more or less impermeable by a covering of concrete, cement, or slate, and from this impermeable layer the rain-water is led away into underground reservoirs by means of pipes. The method of collecting the rainfall off the roof is open to many objections in the Tropics owing to the chances of contamination by dust, excreta of birds, and, it may be added, of insects (*see* "Diarrhœa," *page* 38), and by dead organic vegetable matter. This may be avoided by various contrivances, the objects of which are to reject the first washings off the roof and direct the remaining flow into the tanks. The storage tanks should, as a rule, be made of slate, cement, or earthenware, or simply consist of a tank built of bricks laid in cement. These storage tanks should not be constructed of lead owing to the great absorbing properties of rain-water, and, further, they should be placed upon the ground and covered with mosquito wire netting. It is an advantage to have them covered in, so as to prevent the ingress of insects, rats and dust. They ought at periodic intervals to be cleaned out, and it is essential that the overflow-pipe should communicate with the open air. A further improvement could be made by adding a sand filter which would receive the rain direct from the roof before reaching the vats.

Reference may be made to a very common source of water supply in the Tropics, viz., wells. As a rule, three types of wells are met with—shallow, deep, and artesian wells—and a reference to the use and abuse of these wells in the Tropics might well be made here. Shallow wells may vary in depth from two to fifty feet, and they derive their water supply from the sub-soil water. The chief objection to shallow wells is the readiness with which they become polluted from cesspools and surface drainage, which form the commonest sources of contamination in the Tropics.

In some parts it is not an uncommon sight to see natives performing the act of ablution at the mouth of a surface well, and it does not require much imagination to see how such a well becomes contaminated. Further, the proximity of the well to burial-grounds, cesspools and other filth accumulations, does not improve its chances of escaping contamination. It is essential, if surface-wells are to be used, that the ground should be quite clear for a radius of at least 100 feet, and the ground water level should be not less than twelve feet from the surface.

William Dawson,[3] in an interesting paper on the supply of drinking-water in India and its connection with sub-soil water, suggests that much might be done to further improve the

[1] Mason, E. (1906), "Filaria in the Blood of Camels in Egypt." *Journal of Comparative Pathology and Therapeutics*, p. 118.

[2] Simpson, W. J., etc. (May 1st, 1903), "Water Supplies." *Journal of Tropical Medicine*, p. 132.

[3] Dawson, A. W. (January 1st, 1907), "The Supply of Drinking Water in India, and its Connection with the Sub-Soil Water." *Journal of the Royal Institute of Public Health*, p. 33, Vol. XV., No. 1.

Water— *continued*

health of Indian stations by paying more attention to the geological formation of the ground in the vicinity of the station, and especially to the origin, course and protection of the sub-soil water. He further suggests that a contour map of the sub-soil water should be made by an engineer, and that information regarding the rise and fall of sub-soil water and other necessary geological data should be collected at every station and the necessary maps prepared, so that if any building or entrenching is to be done, the direction of the flow of the sub-soil water will be known. Entrenching of grounds should always be carried out on the downstream side of the sub-soil water supply to a station. Shallow wells are more commonly used than any other variety, and as they draw their supply of water from the sub-soil water, certain conditions are absolutely essential before the sub-soil water can be trusted. (1) the locality should not be thickly populated; (2) the refuse disposed of must be spread over wide areas and not placed in deep pits; (3) there must be a *living* surface, and on no account must this be removed; (4) the porous filtering soil should be of sufficient thickness. It is important also to know the character of the filtering media. If of a sandy nature, purification is greatly assisted by oxidation; but if, on the other hand, the sub-soil consists of clay, purification is practically impossible, as the clay virtually acts as a culture medium, especially in the case of a sub-soil consisting of black clay. The formation of fissures and the presence of disused wells, which are frequently used by natives for insanitary purposes, are other factors which constitute sources of pollution for a water supply.

The ever-present difficulty of surface well contamination in the Tropics can be surmounted by the use of tube or Abyssinian wells. The tube well consists chiefly of an iron tube of a diameter varying from $1\frac{1}{4}$ to 4 inches, which has at its lower end a steel point for boring purposes. Above the point the tube is perforated for some distance to admit water. By means of a weight attached to a tripod the tube well is driven into the ground till water is reached. The upper end of the tube is so constructed as to allow of another tube being coupled on to it. Whenever water is reached a pump is attached. The tube well may tap the ground water overlying the first impermeable stratum, or it may sink lower than this so as to reach a second water-bearing stratum. The bore well has the advantage over the tube well in rapidity in sinking. Deep wells and springs usually provide a pure water, as the filtration through the soil has been so complete as to render the water free from organic matter.

Wells should always be efficiently protected, and should be lined and cemented. The area of the ground around the mouth of a well should be concreted with a slope away from the well, and a raised parapet can with advantage be built so as to prevent surface-drainage flowing in at the top. The suction pipe should be placed at the side of the well, not immediately over it.

Koch[1] devised a method of converting a shallow well into a tube well, and thus protecting it from contamination by means of an iron pipe reaching from the bottom to the top of the well, and by filling up the well to the highest water point with pebbles and gravel and the remainder to the surface with sand.

Various methods have been employed for the detection of suspected sources of contamination, and certain chemical substances are used at the suspected source of pollution and afterwards looked for in the contaminated waters. As a control, it is necessary to examine the water first to ascertain that there is not naturally in it any of the substance to be employed in the test.

The chief soluble chemical substances employed are fluoresceïn, lithium chloride and common salt. The presence of lithium chloride in the contaminated water is detected by spectroscopic examination, that of common salt by means of the silver nitrate test, while fluoresceïn gives a green fluorescence in the presence of an alkali. Dr. Beam, of the Chemical Laboratory, Gordon College, has introduced a new method of detecting the slightest trace of fluoresceïn, an account of which is given in his report. As regards the use of fluoresceïn as an agent for the detection of pollution of wells, if a connection can be established by means of fluoresceïn between a spot known to be contaminated and the source of water supply, such supply should be considered as dangerous and liable at any time to give rise to a water-borne epidemic.

McCrae and Stock[2] conducted a series of experiments in connection with the use of fluoresceïn in South Africa. The method employed by them was to place fluoresceïn in the

[1] Koch, R. (July 22nd, 1905), "Annotation." *Lancet.*

[2] McCrae, J., and Stock, P. G. (April, 1907), "Experiments with Fluoresceïn." *Journal of Hygiene*, p. 182.

Water—*continued*

suspected area of contamination and to pump the neighbouring wells with a view to detecting the presence of fluoresceïn. These observers frequently found that, by ordinary examination, fluoresceïn could not be detected unless the fluoresceïn-containing sample was first concentrated by boiling and the remaining deposit filtered off.

McCrae and Stock state that for the recognition of fluorescence caused by fluoresceïn, it is preferable to examine the solution against a dark background rather than against a white one, and that the use of magnesium light is unnecessary. Further, they found that the addition of an alkali to the fluoresceïn was essential, as in acid solution fluoresceïn does not give a characteristic green fluorescence. Copeman states that fluorescence is appreciable in a dilution of 1 in 100,000,000, and if the concentration method for detecting the presence of fluoresceïn be used, the delicacy of this recognition can be increased.

McCrae and Stock found that 2½ litres can be concentrated for this purpose, evaporation of the water rendering the detection of fluoresceïn more delicate. They state that concentration should not be carried too far, the best results being obtained when the volume was not reduced below 5 c.c. It is usually necessary to filter the concentrate and to wash the filter paper in 1 c.c. or 2 c.c. of water.

If the water contain iron, care must be taken not to confuse in the concentrate the greenish colour due to the presence of this metal with the green tinge of fluoresceïn. The appearance of the fluoresceïn will be the longer delayed the finer the material through which the water passes.

Lake waters are as a rule very pure, this being chiefly due to the fact that the suspended matters subside, while, owing to the great expanse of water, free oxidation occurs.

The lower forms of plant life, such as *algæ*, are liable to develop, but this difficulty can be got over by screening and filtration. The natural condition of the country surrounding surface waters affects the purity of these waters. The ground around the collecting area should if possible be free from cultivation and should not be inhabited. There is some advantage in having trees planted over the collecting grounds, as they prevent water rushing down in torrents to the lakes and reservoirs, holding it up and allowing of a constant flow.

The system of carrying water from open reservoirs to towns and villages by means of open conduits is one to be condemned as dangerous, owing to the great risk of contamination by the insanitary habits and customs of natives.

Rivers as a source of water supply are not liable to so much pollution in the Tropics as one would imagine, owing to their greater volume, but during a period of heavy rain, they frequently become contaminated by receiving the contents of sewers and the excreta and filth of towns and villages on their banks. Small and shallow rivers are very liable to be polluted, owing to the fact that during the dry season the river beds are liable to become dry and filled up with all kinds of filth and pollution, so that at the commencement of the rains the water in them contains a large amount of filth. It often happens that the river is used for purposes of ablution at the same spot that water is obtained for drinking purposes. This can be avoided by setting aside the part of the river highest up stream for drinking purposes, and that for ablution lowest down.

Ponds and tanks are frequently used, especially in India, as a source of water supply. They are liable to contamination owing to the filthy habits of the natives, periodically become infected with cholera germs, and form a suitable nidus for all kinds of parasites.

In tanks and wells infected with the cholera germ, disinfection by means of potassium permanganate gives excellent results. For an ordinary well four ounces of potassium permanganate is about the net amount required, but more may be necessary. Roughly speaking, the correct amount is that which will colour the well-water pink for half-an-hour. It is an advantage to dissolve potassium permanganate first, before adding it to the well, and a further advantage to add it to the well in the evening.

Lime has also been used for disinfecting purposes, the strength being one ounce to a cubic foot of water.

A new method for the disinfection of drinking-water is described by Paterno and Cingolani.[1] These authors have applied silver fluoride (Tachyol) for the purpose of sterilising water. In a strength of 1 in 400,000 it was found to destroy all organisms except the *B. subtilis* and a few others of no importance, its action being more effectual than

[1] Paterno, E., and Cingolani, M. (October, 1907), "A New Method for the Disinfection of Drinking-Water." *Journal of the Royal Institute of Public Health.*

Water— *continued*

chlorine, bromine or ozone. In water containing in excess salt or organic matter, a strength of 1 in 200,000 is advisable. The addition of Tachyol in a strength of 1 in 500,000 causes a transient turbidity in water, which entirely disappears in 24 hours.

Various methods of purifying water are in use, but mention may be made of precipitation, boiling and filtration.

Of precipitation methods, tannin and the juices of various vegetables have been employed, but the agents most commonly used are alum and lime. Alum, in a strength of 6 grains to the gallon, is an excellent clarifier of muddy water. It has also a bactericidal effect on many water bacteria. The addition of 5 grains of lime further enhances the clarifying effect of these agents. Unfortunately, alum has no bactericidal action on such pathogenic organisms as the typhoid bacillus or cholera vibrio.

Boiling as a method of purifying water is excellent, as all organisms are destroyed, and further the amount of hardness in the water is reduced. The main disadvantage of this method of purification is the fact that the water takes some time to cool sufficiently for potable purposes, and, owing to the air being drawn off, the boiled water has rather an insipid taste. To overcome this difficulty, various water sterilisers have been in the market and of some of these mention may be made.

The Lawrence Patent Water Softener and Steriliser is one in which a special system of boiling is employed, whereby the free carbonic acid, which holds in solution the carbonates of lime and magnesia, is driven off and consequently these carbonates are removed from the water and are deposited in a solid form. The boiling is effected continuously, the water as it is passed through the apparatus being heated progressively until it reaches a state of violent ebullition. It is then rapidly cooled and leaves the apparatus only slightly warmer than when it entered it. The smaller plants can deal with from 100 to 3000 gallons per hour, while there are large town supply plants treating 50,000 gallons per hour.

The features which are said to distinguish this system are :—

1. Low first cost and low working expenses.

2. Absolute efficiency and rapidity of action.

3. No chemicals are used, except occasionally a little carbonate of soda, *i.e.* when it is desired to get rid of permanent hardness. Great simplicity and ease of working, no complicated parts, and nothing to get out of order.

4. The utmost ease of cleaning, only one-half the deposit of a lime process, and that in a solid form.

A small apparatus of this type has been recommended for use at the Fort, Khartoum, where the well water is both hard and impure, and where difficulties as regards transport, etc., prevent the river supply being utilised.

There are also the Forbes[1] Water Steriliser and the Naiche[2] Automatic Water Steriliser on the market. Both of these sterilisers claim the advantage that the air of the water is retained in solution.

The Ford-Palliser[3] Drinking Tank is another form of steriliser placed on a cart and intended for use in the army. The advantage of this drinking tank is that 50 gallons of water can be boiled in an hour, while the cart is in motion; the tank is kept level by means of a gimballed arrangement and the water is cooled by a tank containing saltpetre. Faichnie[4] calls attention to an important point in connection with water supply for troops in camp and on the march, viz., the great importance of a pure water supply for cleansing vessels used for eating and drinking. If boiling water be not available for washing up plates and knives and drinking vessels these may all become infected by the use of unsafe water, and the virus may remain active for days. Methods of water analysis always require time and are quite impracticable in camp and on the line of march, but the difficulty of supplying pure water to the troops is now more or less surmounted by the use of the new pattern of army cart, which is fitted with sponges to stop sediment and with candles to stop microbes, and can supply 210 gallons of water per hour. Where there is difficulty in using carriage transport, as in mountain warfare, mule filters are found to be very serviceable.

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[1] Simpson, W. J. (June 1st, 1903), "Water Supplies." *Journal of Tropical Medicine*, p. 172.

[2] Simpson, W. J. (June 15th, 1903), "Water Supplies." *Journal of Tropical Medicine*, p. 192.

[3] Ford-Palliser Drinking Tank. *Lancet*, June, 1904.

[4] Faichnie, N. (August 31st, 1907), "Water Supply in the Camp, on the March, in Battle." *British Medical Journal*.

Water—*continued*

When the soldier is some distance from transport he must be supplied with means of readily obtaining pure water, and the use of Vaillard's red, white and blue tablets or syniodules is recommended. These consist respectively of—(1) Potassium iodide together with sodium iodide (blue); (2) tartaric acid (red); (3) sodium hyposulphite (white). To purify a litre (nearly two pints) of water, dissolve, simultaneously and completely, one blue and one red tablet in two or three tablespoonfuls of cold water. A yellow-brown liquid is obtained. Add this to the litre of water to be purified. Shake and mix well. After 10 minutes add one white tablet, and the yellow water, after shaking, becomes colourless and drinkable. The action is due to the liberation of free iodine which, in a dose of 25 mm. to the litre of water, kills with certainty, in 5 to 10 minutes, the *B. typhosus*, *B. coli* and *V. choleræ*. In the above operation 60 mm. of iodine are produced. If the tablets were dissolved in the whole quantity of water to be sterilised, the amount produced would be much less, hence it is essential to carry out the operation as above described.

Tablet No. 3 (white), transforms the iodine into iodide, and the quantity of the latter is so small that it exerts no injurious influence. These syniodules are prepared by MM. Lépinois and Michel, 7, Rue la Feuillade, Paris; and can be obtained from A. Lewino, 6, Castle Street, Falcon Square, London, England.

Evan's sterilising tablets, prepared according to Nesfield's[1] process, are also useful for sterilising drinking water, and their action consists also in the free liberation of iodine and its ultimate transformation. (Evan's sterilising tablets can be obtained from Evans, Sons, Lescher and Webb, 60, Bartholomew Close, London, England.)

The bactericidal action of Nesfield's tablets was investigated by Windsor,[2] who found that the addition of one 2-grain tablet of mixed iodide and iodate of sodium, and one similar tablet of citric acid to 4 gallons of water previously sterilised will kill in one minute typhoid and cholera microbes when these are added and are present in numbers exceeding 50,000 per c.c.

Chloros,[3] a commercial preparation of the hypochlorite of soda, was used extensively for the sterilisation of the Lincoln water supply. Used in a strength of 1 in 50,000, it was found to render the water free from objectionable micro-organisms, and at the same time was devoid of harmful properties to the consumer.

The action of Chloros depends on the liberation of oxygen, which in its nascent form acts as an oxidising agent. For the water supply of large communities, purification by filtration is extensively employed, but as this system is not used to any great extent in the Tropics, it will only be necessary to refer to the more important points in relation to filtration methods. The ordinary filters for public supplies consist of water-tight basins of varying depths, with sides and floor built of cement. In the floor are channels for collecting the filtered water. The filter normally is about 5 or 6 feet in depth, and is built up from the bottom with stones or pebbles covered by a layer of coarse gravel on which is placed a layer of coarse sand and finally a layer of fine sand. The water rests several feet deep on the filter surface, and should not be allowed to flow through it at a greater rate than 4 inches per hour. The purifying action of a sand filter depends on the slimy deposit which occurs on its surface. This deposit is composed of finely-divided clay, with powerful absorbent properties, and a gelatinous mass consisting of bacilli, streptococci, algæ and other organisms which have been intercepted. Immediately below this slimy deposit is a layer of nitrifying organisms. The formation of this vital layer on the filter may take any time from three to twenty-four hours, and during this period the water which passes through is not free from impurities and should be allowed to run waste. It is essential to fill the filter-bed from the top, and the water should be allowed to stand in it at a depth of 3 feet for at least twenty-four hours.

Sand filters require constant supervision, otherwise the effects of purifying the water are nullified and the filters become a source of danger. Filters ought to be covered to prevent the possibility of their becoming frozen in cold climates, and in hot climates to prevent the water becoming too warm and favouring the growth of algæ. When the slimy layer becomes too thick for the water to pass through, it should be removed. The filter

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[1] Nesfield, V. B. (October, 1905), "On the Sterilisation of Drinking-Water by the Liberation of Free Iodine." *Journal of Preventive Medicine.*

[2] Windsor, C. F. (August, 1905), "The Bactericidal Power of Nesfield's Method of Purifying Water, *Indian Medical Gazette.*

[3] "The Disinfecting Value of Chlorine." *Public Health Engineer.*

Water— *continued*

should be cleaned at regular periods by removing a thin layer of sand half an inch in thickness and disturbing the upper part of the remainder of the sand by means of a fork or rake, so as to expose it to the air. This process is repeated till the upper layer of fine sand is reduced to a foot in thickness, after which the whole filter is cleansed. Koch maintains that for efficient filtration the sand should not be below a foot in thickness, the rate of flow should not be more than 3·95 inches per hour, and the number of microbes should not exceed 100 per c.c. in the filtered water.

There are two systems of sand filtration—the slow and the rapid. In the former, the process is dependent on the gravitation of the water through the filter; in the latter, by the aid of mechanical contrivances, the rate of filtration is generally about fifty times as great as that of the ordinary gravity filter. In these mechanical filters, an artificial film of an intercepting nature is obtained by the addition of some substance, usually sulphate of alumina, and they are, as a rule, in covered vats or tanks and subjected to pressure by the admission of compressed air. For domestic purposes the Pasteur-Chamberland and Doulton filters are useful, as they render the water bacteria free. Asbestos, charcoal and similar filters now possess only an historical interest as they are quite inefficient. The utility of Pasteur-Chamberland filters is greatly inhibited in the Tropics owing to the frequent muddy condition of the water, which completely blocks up the filter, hence necessitating frequent cleansing. This may be overcome to a certain extent by clarifying the water prior to filtration.

In the *Union Pharmaceutique* a simple and apparently harmless method is described for purifying potable water.[1] The method, as described by M. Celestion Hy, is based on that originally devised by MM. Gerard and Bordes. The water to be purified is first treated with a powder consisting of one part potassium permanganate to seven parts sodium carbonate and slaked lime. After five minutes interval, eight parts of anhydrous ferrous sulphate are added. The method is based upon the fact that potassium permanganate in an alkaline solution oxidises organic matter and destroys micro-organisms. The sodium carbonate precipitates any calcium sulphate that may occur naturally in the water, and the calcium hydrate precipitates any bicarbonate of lime that may be present. On adding ferrous sulphate, the excess of permanganate is removed in the form of a dense precipitate. The water drawn off from the precipitate is pure and limpid and contains only a small amount of the sulphate of potassium and sodium. Their presence is not at all objectionable and there is very little danger in using an excess of either of the powders. Howard Jones,[2] in an interesting paper, describes a method of efficiently removing from reservoirs the objectionable smell caused by the presence of algæ in the water supply for Newport. Having noted the results obtained in America by the use of copper sulphate for the removal of algæ, it was decided to employ copper sulphate. Ten pounds of copper sulphate were used for every million gallons of water, the copper sulphate being towed in bags behind a raft. This treatment was successful in the course of a few days.

With regard to the value of storage as a method of purifying water, Houston[3] maintains that the chief importance of storage lies in the fact that the micro-organisms of water-borne disease gradually die in the struggle for existence when they have to contend against the ordinary water bacteria. If water is stored for weeks and months, the probability of any harmful bacteria surviving is excessively remote, and, if stored sufficiently long, is incapable of giving rise to epidemic disease. Accordingly the Metropolitan Water Board of London opened two new reservoirs for the storage of water, which would therefore pass through a sedimentary stage before reaching the filter beds. Reference may be made to the presence of Crenothrix in a sample of water which was taken from some wells sunk in the vicinity of Khartoum. A note on *Crenothrix polyspora* (Cohn), is given by Rullmann,[4] who found it in the reservoirs supplying water to the town of Landshut, in lower Bavaria. He obtained micro-photographs of this alga stained with carbol-fuchsine.

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[1] (April 4th, 1905), "The Purification of Potable Water." *Lancet.*

[2] Howard Jones (January 1st, 1907), "Copper Sulphate Treatment of Reservoirs." *Public Health*, p. 244, Vol. XIX., No. 4.

[3] Houston, A. C. (June, 1907), "Sedimentation in the Purification of Water." *Public Health*, p. 558, Vol. XIX., No. 9.

[4] Rullmann, W. (December, 1907), Photogramme von Crenothrix Polyspora, Cohn. *Cent. für. Bakt.*, II. Ab., Bd. XX., No. 4/5.

Houston,[1] who has been conducting research work in London, summarises his conclusions regarding the effects of storage on water as follows:— **Water—** *continued*

(*a*) It is most desirable that the question of storage should be looked at from a general standpoint, so as to render the length of time during which water is stored more uniform throughout the different districts; hence the policy of intercommunication, already being applied to the filtered water, should be extended to the stored water as far as this is practicable.

(*b*) The advantages accruing from even a few days' storage may be so material that, exceptional cases apart, the use of *raw* unstored water for filtration purposes should strongly be deprecated.

(*c*) Although, as a counsel of perfection, the water should possibly be stored for one or two months, storage for four weeks may perhaps, in the present state of our knowledge, be regarded as affording a sufficient margin of safety.

(*d*) It is possible to determine, with reasonable accuracy, whether the water being used for filtration purposes has been stored antecedently for such a length of time as to give relative (if not absolute) assurance that any harmful properties it may originally have possessed have been destroyed in the process of storage.

(*e*) It is not impossible that the additional "safety" conferred by adequate storage may come to be regarded as a reasonable pretext for filtration through mechanical filters, at specially rapid rates, thereby effecting considerable economies in the cost of filtration, as ordinarily practised; but any departure from old-established filtration custom should not be entertained in the absence of convincing experimental proof of the reliability of the new process.

(*f*) The question of storage is one both of quality and quantity, and, strictly speaking, the number of days it is *desirable* to store water to improve its *quality* should be added to the minimum number of days of storage, which it is necessary to provide in guarding against the possibility of a shortage of water; nevertheless, during a considerable part of each year, there is an abundance of water of relatively good quality in the Thames and the Lea, and the existing storage reservoirs are sufficiently large *in the aggregate*, to improve enormously the water derived from these rivers.

(*g*) . . . (*h*) The advantages accruing from adequate storage of water are of a general character and are not limited to the elimination of danger from typhoid fever.

Weil's Disease. This comparatively rare but interesting disease known under the various synonyms of febrile jaundice, infectious jaundice, epidemic jaundice, Griesinger's disease, bilious typhoid, is an acute infectious disease characterised by fever, jaundice, enlargement of the spleen and liver, nephritis, and various nervous symptoms. Although occurring in epidemic form, especially in the summer months, it is not a contagious disease. Its geographical distribution is of interest. In Smyrna it has been more or less endemic since the year 1837, and its recognised presence in Alexandria dates from the year 1870.

Griesinger in Cairo, in 1851 and 1852, called attention to its peculiar features, and differentiated it from yellow fever and bilious remittent fever. At the Kasr-El-Ainy hospital, 132 cases were treated by him, and, as a result of various post mortem examinations on cases which died from this fever, he chose the synonym of Bilious Typhoid, owing to the fact that so many organs were affected.

In 1886, during four months, 185 cases were reported from Nauplia in Greece, and Professor Weil, of Heidelberg, published in 1886 four cases of acute infectious jaundice with swelling of the spleen and nephritis, and since then several cases have been recorded from Germany.

Small epidemics have occurred in England, the United States and China, and more or less doubtful cases have occurred in several towns in Egypt, Greece, Malta, Dalmatia, Syria and the Ionian Islands. Larrey's "yellow fever" in Cairo, in 1800, was probably a form of this disease; and in the *British Medical Journal* of 1898, Colonel Crombie noted, amongst the unclassed fevers of hot climates, an outbreak occurring in the Central Provinces of India which was reported by a native doctor as "yellow fever."

Anderson[2]* reported an epidemic in Buxar Central Gaol in India, where sixteen cases occurred, and undoubtedly it may occur in other parts of India and other tropical countries, though described under other headings.

This disease generally attacks men between the ages of twenty and thirty, having a tendency to affect natives and Greeks, though cases have occurred amongst other Europeans. The disease is not contagious, but one attack seems to confer immunity. It is not confined entirely to the poorest classes, for many cases, in an epidemic at Alexandria, occurred amongst professional men, and its seasonal incidence seems to reach the maximum between the months of April and October.

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[1] Houston, A. C. Special Report reviewed in *Lancet*, July 25th, 1908.

[2] Anderson, S. (September 17th, 1904), "Epidemic Catarrhal Jaundice." *British Medical Journal.*

* Article not consulted in the original.

According to Sandwith,[1] this disorder is undoubtedly a filth disease, engendered by contamination with sewage and putrid meat. In Alexandria it has become more common since the introduction of a bad system of drainage, and the bulk of the patients affected came from the lowest parts of the town where the sewers empty into the sea; while the suburbs of the town, which are not drained at all, remain apparently unaffected.

The cause is still obscure, no microbe having yet been described. There is no direct evidence that the disease is insect-borne. Sandwith[2] is inclined to think that either *Culex fatigans* or the *Stegomyia fasciata* is a likely culprit in transmitting it. Taeger and Nauwerk, in Germany, believe that the *Bacillus proteus fluorescens* is the cause of the disease, owing to the fact that this bacillus was isolated from the urine in cases after death had occurred, and because the bacillus was found in ducks and geese which had died in the same locality from a disease in which jaundice was the chief symptom.

The symptoms may be divided into three stages: 1. Primary fever, lasting three to five days. 2. Jaundice, about seven to nine days. 3. Secondary fever, lasting about seven to nine days. After an incubation period of one or two days, the disease is ushered in by a rigor, general pains and vomiting and a temperature of 102° to 104°. About the third or fourth day, jaundice begins with great enlargement of the liver and spleen together with tenderness. Albuminuria also is present, together with a certain amount of suppression which may increase till uræmia supervenes. The fever subsides, jaundice and other symptoms disappear, and in the majority of the cases this improvement is followed by a secondary fever.

The nervous symptoms consist for the most part of headache, giddiness and perhaps delirium, the patient passing more or less into the "typhoid state." Muscular pains, especially in the nape of the neck and the calves of the leg, are intense during the first stage of the disease, and are greatly increased by pressure, this forming a useful diagnostic sign.

The chief complication is that of hyperpyrexia, and it is observed that the convalescence is invariably a protracted one.

The mortality occurs chiefly in those above the age of 40, and may vary from 10 per cent. to 60 per cent.

The chief pathological conditions present consist of an enlargement of the liver with fatty degeneration and concomitant cloudy swelling and infiltration of the portal canal with lymphocytes. The spleen is only slightly enlarged. Petechial hæmorrhages are frequently present in the pleura, peri- and endo-cardium, and capillary hæmorrhages are present in the stomach and kidneys. Microscopically the kidneys show a lymphocyte infiltration around the glomeruli.

Formerly there was some difficulty in diagnosing this disease from relapsing fever, but that difficulty no longer exists, owing to the presence in the latter of a spirochæte. It has been suggested that this disease resembles yellow fever, but, as the latter has not been known to exist in Egypt, infectious jaundice can hardly be confused with it, although some observers state that it is a modified form of yellow fever. Its similarity to acute yellow atrophy must also be borne in mind.

Epidemic jaundice occurred in South Africa during the late war, but Mathias[3] considered that it was a distinct variety of febrile jaundice which frequently follows outbreaks of enteric fever, and that it should not come under the category of Weil's disease.

The treatment is chiefly symptomatic. Bryce-Orme[4] records what appears to have been a fatal case of infectious jaundice in the Federated Malay States, showing therefore that the disease has probably a wider geographical distribution than is supposed, and is not merely confined to the Mediterranean Basin. This observer considered the case to be one of infectious jaundice, the predominating signs and symptoms being intense jaundice, enlarged liver, and albuminuria. There were no indications of the malarial parasite being present in the blood, but there was present a well-marked leucocytosis.

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[1] Sandwith, F. M. (January 15th, 1904), "Weil's Disease in Egypt." *Journal of Tropical Medicine.*

[2] Sandwith, F. M. (September 17th, 1904), "Infectious Jaundice." *British Medical Journal.*

[3] Mathias, H. B. (September 17th, 1904), "Jaundice in South Africa." *British Medical Journal.*

[4] Bryce-Orme, W. (February 29th, 1908), "A Fatal Case of Infectious Jaundice in the Federated Malay States." *British Medical Journal.*

Whooping Cough. Notwithstanding the large number of investigations in connection with this disease the etiology of whooping cough has not as yet been determined. That it is an undoubtedly infectious, transmissible disease is more or less recognised owing to its frequent epidemic character and its endemicity in certain cities; furthermore, it has a definite incubation period. It occurs in the Sudan, but is not very common.

According to Ager[1]* the disease certainly spreads by contact between children, and Baginsky states positively that he has observed transmission by a third person. The virus seems to adhere to rooms and furniture, and one attack usually confers immunity. The literature on the bacteriology of this disease is as confusing as it is extensive, the most striking feature being the lack of unanimity in the results.

Spengler[2]* was the first to describe an organism in pertussis sputum closely resembling the influenza bacillus in its morphological and biological aspects.

Czaplewski and Hensel[3]* found a small, short, polar-staining bacillus slightly larger than the influenza bacillus but which grew upon non-hæmoglobin media. This organism was isolated from sputum on blood-serum plates. Morphologically, it resembles very closely the influenza-like organisms of Spengler, Jochmann and Vincenzi; Czaplewski's results were confirmed by Zusch. Davis[4] examined the sputum of 61 cases, and his observations point to the fact that there is almost constantly present an organism which culturally and morphologically is identified with the influenza bacillus occurring in greater numbers than any other organism. This organism was not abundant during the spasmodic stage of the disease. It has been found several days before the whoop began, and as long as six months after the disease.

There is not, however, sufficient evidence for or against the specificity of this organism for whooping cough. Bordet and Gengou found in pieces of membrane brought up by patients during coughing paroxysms, very numerous delicate ovoid bacteria usually in pure culture, together with numerous leucocytes. These bacteria were sometimes tapering and sometimes so short as to resemble cocci. They were Gram-negative. In favour of the causal relationship of the micro-organism it is to be noted that well-marked agglutination occurred with sera of convalescents from the disease but not with the sera of healthy persons. Similarly, the phenomenon of the deflection of complements was shown in a very high degree by this microbe.

Jochmann and Krause, in 1901, found influenza-like bacilli in the sputum of pertussis cases. These organisms belonged to three different classes, A, B and C, as determined by their reaction to Gram's stain and by their biological properties. Class A contained non-Gram-staining, influenza-like bacilli, growing only in the presence of hæmoglobin; this they called the *B. pertussis eppendorf*. In four cases they found similar bacilli which, however, grew on hæmoglobin-free media. They considered this organism the same as that described by Czaplewski and Hensel. Class C contains a Gram-staining bacillus growing without the presence of hæmoglobin. Magerims believes that the disease is due to a bacillus growing on the Schneiderian membrane of the nose.

With all these observations the proof that these different organisms are the true cause of the disease is still wanting. It is of interest to note that influenza-like organisms have been found in normal throats, and also in several other diseases, amongst which may be mentioned measles, epidemic cerebro-spinal meningitis and varicella. In these diseases influenza-like organisms have been isolated in pure culture from the sputum and from throat swabs.

Macewen[5] successfully conveyed the virus to a healthy cat by feeding it with milk containing the sputum and vomited material of typical pertussis cases. The cat developed whooping cough 17 days afterwards. This experiment appears to show that cats may be the means of disseminating the disease. According to Arnhein, who has made numerous post mortems, there is a marked desquamative catarrh of the larynx and trachea, with swelling of the neighbouring lymph glands. In the diagnosis of pertussis in its early stages,

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[1] Ager, L. C. (November, 1905). *Brooklyn Medical Journal.*

[2] Spengler, C. (1897). *Deutsche Med. Woch.*, 23, p. 830.

[3] Czaplewski, E., and Hensel, R. (1897). *Deutsche Med. Woch.*, 23, p. 586.

[4] Davis, D. & J. (March 2nd, 1906), "Bacteriology of Whooping Cough." *Journal of Infectious Diseases.*

[5] Macewen, H. A. (January 18th, 1908), "The Conveyance of Whooping Cough from Man to Animals by direct Experiment." *British Medical Journal.*

* Article not consulted in the original.

Whooping Cough— *continued*

Churchill states that a differential leucocyte count of the blood is very useful. In most cases there is a marked leucocytosis, and in the early catarrhal stage, when infection is apt to spread, there is a lymphocytosis. Ager mentions, as important diagnostic features, apart from the characteristic cough, the presence of an ulcer on the *frenum linguæ* and the occurrence of a heavy white precipitate of uric acid in the urine.

Parkinson[1]* mentions that whooping cough may be simulated by fibrosis of the lung. As regards treatment, many drugs are in the market, but no specific has as yet been discovered either to abort or prevent an attack.

Mention may be made of a vapour used as an inhalation recommended by Kraus,[2] consisting of naphthalene 180 parts, camphor 20 parts, eucalyptus oil 3 parts, and pine oil 3 parts. This is mixed with boiling water to vaporise it.

Beryaete strongly recommends the use of warm baths at a temperature of 102°-107° F., repeated every 6 hours.

Stephens[3]* recommends syringing the ears with warm boracic acid lotion and painting the throat with 5-10 per cent. solution of cream in glycerin and water.

Rothschild[4]* recommends chloroform narcosis as a successful line of treatment. Various antispasmodics, asafœtida, bromides, antipyrine and belladonna may be tried together with efforts to improve the general health of the patient.

Yaws. This interesting disease has an extensive geographical distribution in many parts of the Tropics. It is especially common in many of the islands in the West Indies, the West Coast of Africa, and occurs in Ceylon and in many of the Pacific Islands, notably Fiji. It is also prevalent in Assam and Burma.

Its nomenclature is as interesting as its distribution, as it is known under the names of Frambœsia, Paranga (Ceylon), Puru (Malay), Pian (Indo-China), Coko (Fiji).

Little is known at present of its distribution in India; its existence there has been confirmed, but its exact extent requires investigation.

It is due to the efforts of Castellani[5] that light has been thrown upon the etiology of yaws; for it was in February, 1905, that this observer announced his discovery of a delicate spirochæte in the secretion obtained from an ulcer in a case of yaws. In a paper[6] read before the Ceylon branch of the British Medical Association, he named this organism the *Spirochæta pertenuis*. Castellani's observation was confirmed by Wellman,[7] Powell and others.

In further observations made by Castellani,[8] spirochætes showing differences in morphological details were noted in the ulcerative lesions of yaws. These spirochætes were of a coarser variety and were termed *S. obtusa* and *S. acumina*.

It is beyond the scope of this work to refer to the minute histological characters of the *Spirochæta pertenuis*, but mention may be made of the difficulty experienced in staining it. By using, however, Leishman's method and allowing the alcoholic solution to act on the film for five to ten minutes, and the subsequent admixture with distilled water to act for half-an-hour to several hours, good staining effects can be obtained. Giemsa's stain also gives good results.

In preparations obtained by Lœffler's method of staining for flagella, Castellani observed in some parasites the presence of an extremely delicate flagellum at one end, and he considers therefore that the organism should be considered as a *Treponema* instead of a spirochæte. That the *Treponema pertenuis* is the causative factor of this disease is now well established. Neisser, Baermann and Halberstädter[9] showed that monkeys could be

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[1] Parkinson, A. S. (August, 1906). *Sediater*, p. 502.

[2] Kraus, E. (1905). *Deutsche Med. Zeit.*, p. 827.

[3] Stephens, T. (1906). *Hospital.*

[4] Rothschild, H. de (May 23rd, 1906). *Sem. Méd.*

[5] Castellani, A. (August 15th, 1905). *Journal of Tropical Medicine.*

[6] Castellani, A. (June 17th, 1905). Paper read before Ceylon Branch of British Medical Association.

[7] Wellman, F. C. (December 1st, 1905). "A Spirochæte found in Yaws Papules." *Journal of Tropical Medicine.*

[8] Castellani, A. (November 23rd, 1907). "Notes on the Spirochæte of Yaws." *British Medical Journal.*

[9] Neisser, A., Baermann and Halberstädter, L. (July 10th, 1906). "Researches on the Transmission of Yaws to Monkeys." *Munch. Med. Woch.*, Vol. LIII., p. 1337.

* Article not consulted in the original.

successfully inoculated with material obtained from the granulomata of a typical case of yaws and from the glands of the arm of a case in full eruption. In the lower monkeys the first symptoms appeared in twenty-two to ninety-one days after inoculation, and in the anthropoid apes the symptoms appeared in thirteen days. **Yaws—** *continued*

Yaws then can be transmitted from man to monkey, and once in three attempts the authors succeeded in infecting another monkey from the first. Of the internal organs and tissues, only the bone-marrow and the cubital glands gave positive results after inoculation. These observers further noted an important point, namely, that a monkey could be infected with yaws fifteen days after the appearance of a syphilitic chancre, proving that monkeys inoculated with syphilis were not immune to yaws. Castellani,[1] in some further observations, showed that the infection of yaws in monkeys is a general one, spirochætes being found in the spleen and lymph glands. He confirmed the observation of Neisser and others that monkeys successfully inoculated with syphilis do not become immune to yaws, and *vice-versa*; and by means of the Bordet-Gengou reaction he detected specific yaws antibodies and antigen. Further, he proved that the specific yaws antibodies and antigen were entirely different from syphilitic antibodies and antigen, thus adding to the chain of evidence that yaws and syphilis are two entirely different entities.

Ashburn and Craig[2] further support the work of Castellani, and undoubtedly their experiments prove that the *Treponema pertenuis* is constantly present in the lesions of yaws.

Reference may be made to the histological characteristics of the yaws nodules studied by Charlonis, Unna, Glogner and others, including Marshall,[3] who found that the *Treponema pertenuis* causes a colliquative necrosis of the epithelial structures in which the parasite is found in large numbers. Ulceration follows this necrosis, and there is a new formation of epithelium in the form of down growths, which, in time, often degenerate, vascular dilatation, leucocyte infiltration and œdema occurring in the corium. There is no endarteritis. There is a peculiar distribution of the polymorphonuclear eosinophiles, and Glogner demonstrated the presence of giant cells.

That yaws and syphilis are two different diseases has been proved now beyond doubt by the experimental researches of Castellani, Neisser and others, in monkeys, and by the observation of Charlonis, who, in 1881, showed that patients suffering from yaws could be infected with syphilis.

Yaws not only differs from syphilis in its clinical features, but also in its geographical distribution. The most important points bearing on the specific entity of these two diseases may be briefly summarised:—1. In non-ulcerative papules, in the spleen, in the lymphatic glands of yaws, patients as well as in inoculated monkeys, the *Treponema pertenuis* is the only organism present. 2. The extract of yaws material containing the *Treponema pertenuis* is infective to monkeys. 3. The extract of yaws material from which the *Treponema pertenuis* has been removed by filtration becomes inert and monkeys inoculated with it do not contract the disease.

Neisser,[4] while pointing out the directions in which yaws and syphilis resemble one another, decides that yaws cannot be merely a degenerate form of syphilis, because (*a*) Yaws does not protect against syphilis, and (*b*) syphilis does not protect against yaws.

Robertson[5] advances evidence to prove that flies can carry the virus.

The communicability of yaws still requires investigation. It is well known that in most cases yaws is conveyed by direct contact from person to person, usually by absorption of the virus through a pre-existing abrasion or wound on the surface of the skin. Among the natives in Ceylon, the primary lesion frequently develops in women on the skin of the trunk just above the hip, due, no doubt, to their method of carrying their children astride of their hips.

According to Jeanselme,[6] the disease is propagated among the adult natives in Indo-China by the chopsticks used instead of forks, by the water-pipe which is handed from mouth to mouth, and the sleeping mats common to all.

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[1] Castellani, A. (July, 1907), "Experimental Investigation on Framboesia." *Journal of Hygiene.*

[2] Ashburn, P. M., and Craig, C. F. (October 1st, 1907), "Observations on Treponema Pertenuis (Castellani) of Yaws, and the Experimental Production of the Disease in Monkeys." *Philippine Journal of Science.*

[3] Marshall (October 1st, 1907), "A Histologic Study of Yaws." *Philippine Journal of Science.*

[4] Neisser, A. (March, 1908). *Arch. für Schiffs-und Trop. Hyg.*, No. 2.

[5] Robertson, A. (July 15th, 1908), "Flies as Carriers of Contagion in Yaws." *Journal of Tropical Medicine and Hygiene.*

[6] Jeanselme, E. (November 11th, 1905), "Notes on Pian," translated by Sandwith. *British Medical Journal.*

Yaws— *continued*

The rôle of biting-flies in spreading this disease still requires to be investigated. Castellani[1]* has shown that under certain conditions the disease may be spread by flies (*Musca domestica*). Modder[2] suggests that the *Ixodes bovis* might be the transmitting agent, but, in a more recent paper, is inclined to believe that the *Argas* ticks are concerned in the spread of this disease.

As regards treatment, Castellani[3] has found, in obstinate cases, the mixed method of treatment to be best. That observer obtained the most satisfactory results with iodide of potassium, followed either by atoxyl or quinine or sodium cacodylate. The iodide of potassium was administered in doses of 1 gramme in milk thrice daily; the atoxyl by daily subcutaneous injections of 0·05 grammes, and one injection of cacodylate of soda or quinine cacodylate containing 0·05 grammes of the drug. As regards local treatment, the same observer recommends the application of perchloride of mercury 1 in 1000, and dusting the granulomata with iodoform or boric acid. For ulcerative lesions he recommends the use of 20 per cent. protargol ointment.

Campbell, Graham,[4] obtained encouraging results with sodium bicarbonate internally in 4-grain doses, together with the local application of copper sulphate.

Yaws does not appear to be indigenous in the Sudan, but Ensor has reported one undoubted and one doubtful case at Kassala. In both instances children were affected. He suggests that the disease may have been introduced into this part of the Sudan by Hausa pilgrims from the West Coast on the way to Mecca.

(I have seen one case of supposed yaws in a Sudanese soldier at Khartoum. My own opinion is that the disease was syphilis, and it yielded very rapidly to large doses of iodide of potassium. I failed to find *S. pertenuis* in smears from the lesions.—A.B.)

Yellow Fever. It was the excellent investigation of Major Reed and his co-workers which led, in 1900, to the discovery of the chief agent in propagating the virus. They found that a *Stegomyia fasciata*, which had fed on an infected case within the first three days of the disease could, after an interval of twelve to twenty days, infect a non-immune patient and invariably transmit the disease. They further observed that fomites of all kinds, clothing, bedding, etc., played no part in the transmission of the virus, and that the disease could be transmitted by the injection of the blood of patients affected with yellow fever during the first three days of the disease.

Marchoux, Salimbeni and Simond[5] during their investigations at Rio confirmed Reed's observations, and found that the serum of a patient suffering from yellow fever, although virulent on the third day of the attack, was not so on the following day. They observed that the virus will pass through a Chamberland F. bougie. They failed to find, either in the blood of man or in the mosquito, the causative agent of yellow fever, but they favoured the idea that the micro-organism was of the nature of a *spirillum*.[6]*

The *B. icteroides* of Sanarelli, Tombleson's *bacillus* and Finlay's *protozöon* must now be looked upon as matters of historical interest only, for the organism of yellow fever, which has been declared by some to be ultra-microscopic, has yet to be discovered.

As regards the transmitting agent, the *Stegomyia fasciata*, some interesting facts have been elucidated. Marchoux[7] and his colleagues found that the *Stegomyia* was not dangerous till after an interval of at least twelve days had elapsed since its last feed on the virulent blood of yellow fever cases, and that the mosquito bite was much more dangerous later than shortly after the insect acquired infection. Some further investigation by Marchoux and Simond[8]* into the life-history of *S. fasciata*, shows that it can bite man,

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[1] Castellani, A. (July 1st, 1907), "Experimental Investigation on Frambœsia Tropica." *Journal of Hygiene.*

[2] Modder, E. E. (June 1st, 1907, and November 15th, 1907), "Transmission of Yaws by Ticks." *Journal of Tropical Medicine and Hygiene.*

[3] Castellani, A. (November 23rd, 1907), "Observations on the Treatment of Yaws." *Lancet.*

[4] Graham, Campbell (November 11th, 1905), "Notes on Frambœsia Tropica." *British Medical Journal.*

[5] Marchoux, E., Salimbeni, I., and Simond, P. L. (November, 1903), "Report of French Mission at Rio." *Ann. de l'Institut Pasteur.*

[6] (June 15th, 1906), "French Yellow Fever Mission to Rio." *Journal of Tropical Medicine.*

[7] Marchoux, E., and Simond, P. L. (January and February, 1906), "Report of French Mission." *Ann. de l'Institut Pasteur.*

[8] (December 1st, 1906), 2nd, 3rd, 4th "Memoirs of French Mission to Rio to study Yellow Fever." *Journal of Tropical Medicine.*

* Article not consulted in the original.

Yellow Fever— *continued*

either by day or by night, from the first day of its adult life. After a few days, however —particularly after laying her first batch of eggs—the female only bites during the night, and it therefore follows that man becomes infected almost invariably during the hours of darkness. Their experiments on the conveyance of yellow fever by mosquitoes other than *S. fasciata*, proved negative. In most other species of mosquito the female invariably dies immediately after laying her first and only batch of eggs, and this circumstance hardly admits of sufficient time for the virus to develop within her. Marchoux and Simond observed that the female *S. fasciata* was capable, after having bitten yellow fever patients, of transmitting to its offspring the power of infecting other human subjects with the disease; but Rosenau and Goldberger[1]* failed to corroborate this. The French Commission[2]* further notes that the *S. fasciata* requires human blood for the development of her eggs, and that when the blood in dead, infected *Stegomyiæ* is ingested by non-infected mosquitoes, infection of the latter results.

Among the special characters of the *S. fasciata* which affects its rôle as a carrier of yellow fever, Carter[3]* notes the fact that it can be conveyed by vessels indefinite distances, and in greater numbers in sailing vessels than in steam-ships. It appears to have a long life-history, 154 days being recorded as the duration of life of an infected insect; further, it is a domestic mosquito, breeding in cisterns, yards and puddles.

With reference to the longevity of the *S. fasciata* on sailing vessels, a serious question arises in connection with the spread of yellow fever into Asia viâ the Panama Canal. Manson[4]* was the first to direct the public attention to this all-important point, and recommended systematic examination and thorough prophylactic measures adapted towards the destruction of the *Stegomyia* on all vessels passing through the Canal. Although the excellent work of Reed and other observers has most conclusively shown that the *Stegomyia fasciata* is the transmitting agent of the yellow fever virus, it is of interest to note that quite recently Ybarra,[5]* in an epidemic which occurred in Cuba, quotes nine cases in which the disease developed after six days' hypothetical exposure to the supposed mosquito infection, but in reality this mosquito infection, he says, could not possibly have existed, and he therefore disputes the theory that the mosquito is the exclusive carrier of the disease.

Various experiments, too numerous to be mentioned here, have been carried out in connection with the virus of yellow fever. Marchoux and Simond found that the virulent serum lost its efficacy at a temperature of 55° C., a point which was previously observed by Reed and others, and that the serum loses its power of infection after exposure for 48 hours to the air; further, if kept at a temperature of 24°–30° C., it is powerless at the end of 48 hours.

Attempts by Marchoux and Simond to infect animals with yellow fever by submitting them to the bites of infected *S. fasciata* failed. Wolferstan Thomas,[6] however, succeeded in infecting a chimpanzee with a benign attack of yellow fever, when infected *Stegomyiæ* were allowed to feed on it.

The incubation period of yellow fever is usually three days, sometimes five, and in one authenticated case six days, but the French observers, Marchoux and Simond, extend it to thirteen. It is beyond the scope of this Review to discuss the symptoms and treatment, but mention may be made of the success obtained by Sternberg's method of treating his cases with alkalies, and, judging from statistics, this method augurs well. The question of prophylaxis is an important one, and one which should be carried out vigorously from the outset. Non-infected *Stegomyiæ* should be prevented from biting the sick, and infected ones from biting the healthy. To this end all collections of water should be abolished or covered with oil, and all cisterns covered with copper-wire gauze. Early notification is essential.

The patient should be segregated during the infective period and infected mosquitoes prevented from escaping from the room. All doors, windows and apertures should be securely screened with gauze, and so also the patient's bed. While the breeding-places are

[1] Rosenau, — and Goldberger, — (1906), Yellow Fever Institute, Bulletin No. 15.

[2] (June 15th, 1906), "French Commission to Rio." *Journal of Tropical Medicine.*

[3] Carter, — (May 14th, 1904), "Some Characteristics of Stegomyia Fasciata, which affect its conveyance of Yellow Fever." *Medical Record.*

[4] Manson, P. (March 7th, 1903), "Introduction of Yellow Fever into Asia." *Lancet.*

[5] Ybarra, A. M. F. (October, 1907), "Recurrence of Yellow Fever in Cuba." *Texas Medical Journal.*

[6] Thomas, W. (January 19th, 1907), "Yellow Fever in the Chimpanzee." *British Medical Journal.*

* Article not consulted in the original.

Yellow Fever— *continued*

guarded the adult insects should be destroyed by sulphur or formaldehyde fumigation, which should be carried out efficiently. (*See* "Mosquitoes," *page* 135.) For the fumigation of the room, pyrethrum burned in the proportion of one pound per thousand cubic feet. An exposure of two hours will stupefy the mosquitoes, causing them to fall to the ground whence they can be swept up and burnt. For the purpose of freeing a ship from mosquitoes, the holds should be fumigated with sulphur and the bilge tanks covered with a film of crude petroleum.

It is essential that shipping should be under rigid inspection. If yellow fever has occurred on board a vessel, the case should be isolated in a quarantine hospital free from *Stegomyia*, and the crew prevented from communicating with the shore for a period of ten to thirteen days.

A point in relation to *S. fasciata* may be noted, viz., the United States Bureau of Entomology have discarded the term *S. fasciata* for that of *S. calopus*.[1*]

Recently new light[2*] has been thrown upon the original discovery of the part played by the mosquito in yellow fever and malaria.

Dr. Aristides Agramonte, in a recent article in the *Cronica Medico Quirurgica de la Habana* on sporadic cases of yellow fever, informs his readers that the credit of being the first observer to call attention to the part played by the mosquito in yellow fever and malaria is due to the late Dr. Beauperthuy; for it was as far back as 1853 that Beauperthuy, writing in the May number of the *Gaceta Official de Cumana*, said that the affection known as yellow fever is due to the same cause as that producing intermittent fever, and that both diseases are caused by the bites of mosquitoes. It is of interest to note how accurate Beauperthuy's observations on yellow fever were in the light of our present knowledge of this disease; and, in fact, his remarks on the subject of fevers in general are remarkably close to the truth.

Unfortunately, Beauperthuy's theories did not receive, at that time, the recognition that was due to them, and it is but fitting and just that this observer should receive the credit of being, so far as at present known, the first to suggest the rôle played by the mosquito in connection with yellow fever and malaria.

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[1] "The American Term for Stegomyia Fasciata" (January 12th, 1907). *British Medical Journal.*

[2] "A Pioneer in Research on Yellow Fever" (May 30th, 1908). *British Medical Journal*, p. 1306.

* Article not consulted in the original.

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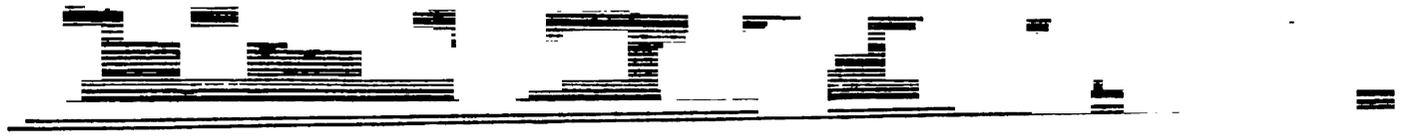

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